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Functional [illegible] Ankylosis of the Stapes — *Samuel Rosen and Max Bergman*

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Effects of Te[illegible] on Hearing Thresholds — *John F. Corso*

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JANUARY [illegible] 1956

VOLUME 63 [illegible] NUMBER 1

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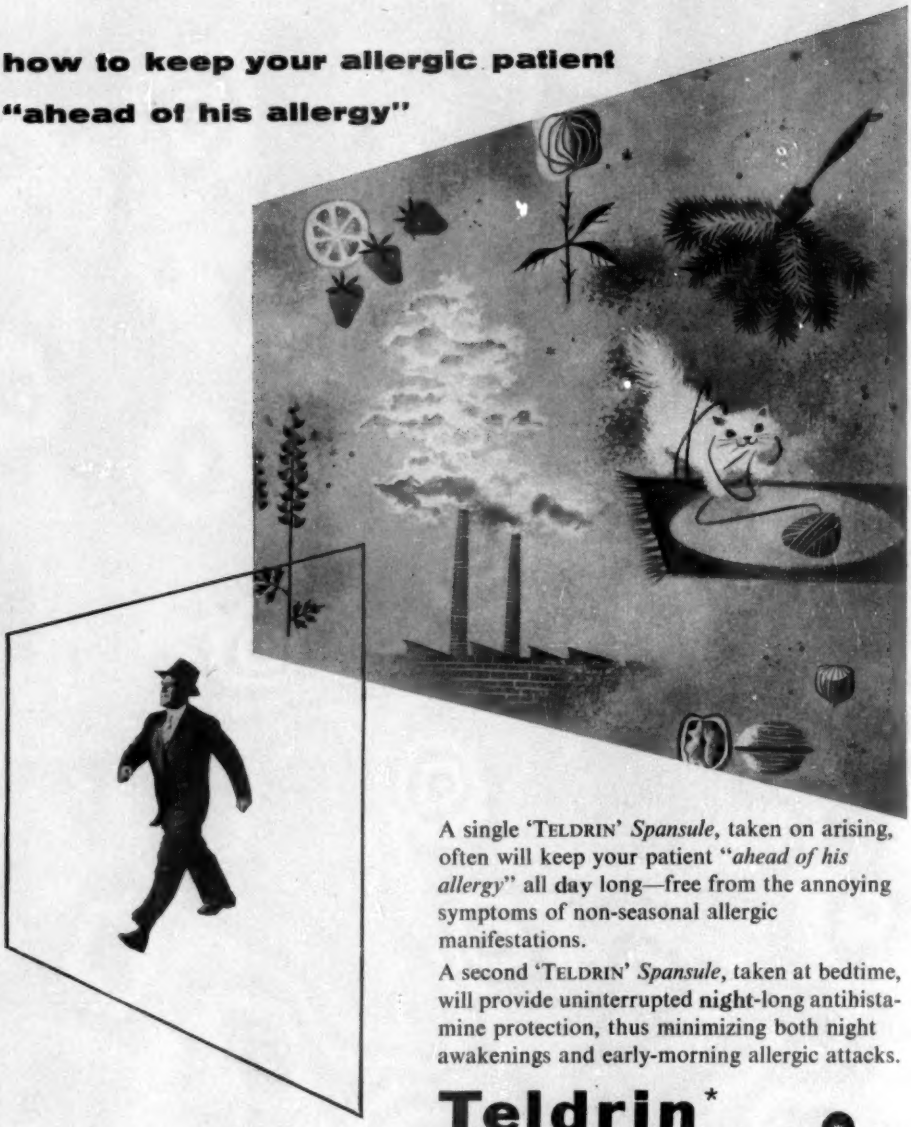

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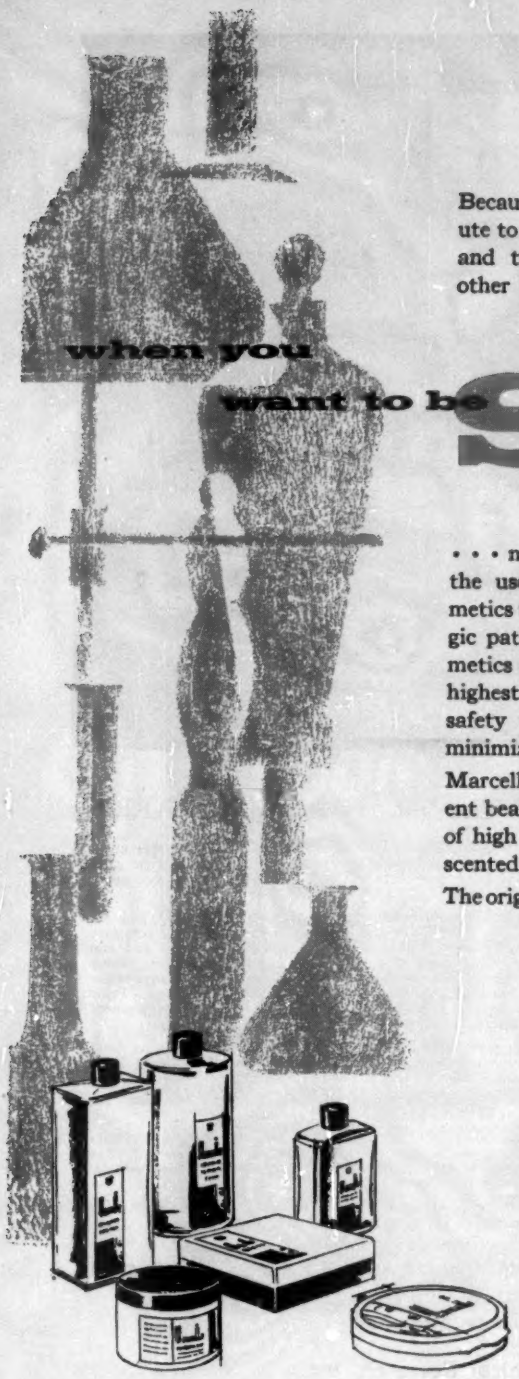

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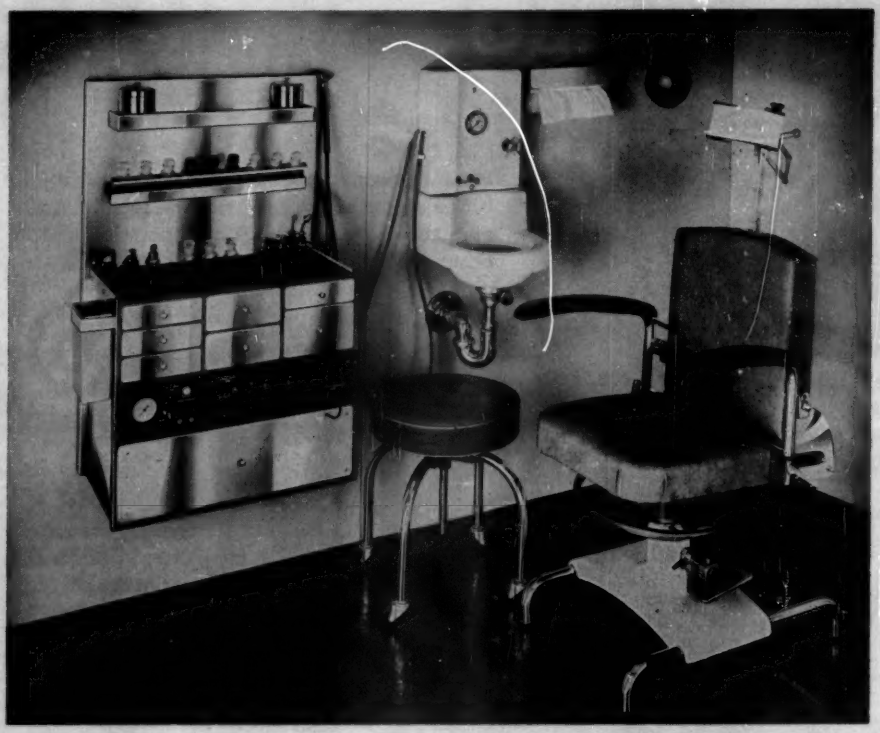

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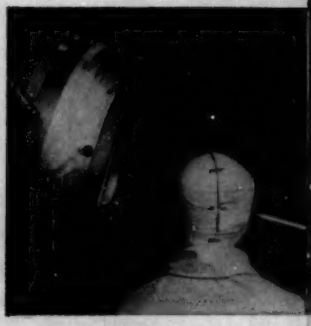

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Editorials

BUILDING YOUR OWN RETIREMENT FUND

An editorial in the April 24, 1954 issue of the *Saturday Evening Post* relates the story of a Philadelphia physician enjoying a large private practice who retired to accept a job in a government hospital, since he found he was unable to educate his children and at the same time save enough to provide for his eventual retirement under the existing tax laws for self-employed individuals. As an employee, he was eligible to participate in a pension plan which would provide him with a retirement income. The *Post* pointed out that a young doctor or lawyer, under our existing tax structure, was likely to find it impossible to put aside savings after taxes to provide for his old age. The professional man, like everyone whose income fluctuates widely over a working lifetime, is the victim of a big tax bite in the most productive years. He has a further disadvantage in that his self-employed status denies him enough from his earnings-after-taxes to finance retirement, or continuing to work long after his employed contemporaries are collecting pension benefits. The exclusion of the self-employed person from the benefits of OASI by his continued earnings after 65 only adds insult to injury.

The Jenkins-Keogh bills are designed to fill the needs of the self-employed, who were left out in the cold when laws were passed permitting a tax shelter for company pension plans. It should be understood that the Federal Internal Revenue Code provides substantial tax advantages to deferred compensation under so-called "approved pension plans," which is not at present matched by similar savings incentives for the self-employed. The Jenkins-Keogh bills provide that "Any individual who is not eligible to participate in a pension or profit-sharing plan may set aside each year an amount not to exceed ten per cent of his earned income, and in no case more than $7,500, to be paid into a restricted retirement trust for insurance annuity. The amount thus set aside could be deducted from his taxable income. The proposed law places certain restrictions on the means by which these savings can be accumulated, and provides that the taxpayer may not tap the fund until he is sixty-five years old, except in the case of total disability."

The American Medical Association is endeavoring to arouse physicians to the advantages of this bill. The legal profession, which finds itself in the same neglected group of taxpayers as physicians, has brought this reform movement close to fruition under the leadership of the American Bar Association. The final destiny of these bills now rests with those individuals who can be counted upon to put their shoulders to the wheel at the appropriate moment. The fall recess finds most congressmen and senators at home, and this is an excellent time for all physicians to let them know of their interest in the proposal, which in amended form is now before the House Ways and Means Committee. If all goes well during the second session of the 84th Congress, it is probable that it will no longer be an economic sin for a man to work for himself.

FRANCIS H. ADLER, M.D.

A.M.A. Archives OF OTOLARYNGOLOGY

Introduction of the Honorary Chairman, Dr. Galloway

FRANCIS W. DAVISON, M.D., Danville, Pa.

William James said, "The great use of a life is to spend it for something that outlasts it." Tom Galloway's medical papers will continue to influence future generations. Most of you are as familiar as I am with Dr. Galloway's many important contributions to the otolaryngologic literature. It was the knowledge of these contributions that prompted your Executive Committee to select Dr. Galloway as the Honorary Chairman of our Section for 1955. Because I wanted to know more about Galloway the man, I wrote to Dr. Soper, one of his associates, for information. The following sentences are quotations from Dr. Soper's letter:

Dr. Galloway is one of the old school who has taken his work very seriously. One of his most outstanding characteristics has been his never-give-up attitude even in the most desperate cases.

He has done a great deal to educate the profession to the need of early tracheotomy in cases of acute laryngotracheobronchitis and bulbar polio. He has kept up a deep interest in other fields of medicine and his judgment is widely respected. This knowledge of other fields has enabled him to use the facts determined by laboratory scientists in the solution of clinical problems.

He has declined some of the honors offered him in recent years because he felt that the work should be taken over by stronger, younger men. I am afraid this is not an adequate description for such a remarkable person, but it will give you a little to start on. He can be very proud of the many young men whose careers have been started by his instruction in scientific medicine and whose ethics have been influenced by his fine philosophy. Many patients are alive today only because of his scientific knowledge and energetic treatment when their lives were threatened by respiratory obstruction.

One of my vivid memories of Tom Galloway is his presentation in 1949 of the historic paper entitled "Bulbar Poliomyelitis: Favorable Results in Its Treatment as a Problem in Respiratory Obstruction."[1] Today his ideas are widely known and accepted, and, as a result, many hundreds of lives have been saved. At that time he still received adverse criticism despite the fact that he was able to report 15 consecutive recoveries in serious cases of bulbar poliomyelitis when the mortality rate for the same area in the same period was 24%. I suspect this one paper has done more than all others to educate the profession to the fact that death in cases of bulbar poliomyelitis is due to cerebral hypoxia, the result of secretional obstruction, and that it can be prevented by postural drainage and early tracheotomy.

Time will permit mention of only a few of Dr. Galloway's outstanding contributions. I select the following because they have been an important part of my education: "Postural Treatment of Acute Laryngotracheobronchitis,"[2] "Tracheotomy in Bulbar

Accepted for publication Aug. 17, 1955.

Chairman of Section on Laryngology, Otology and Rhinology.

Read before the Section on Laryngology, Otology and Rhinology at the 104th Annual Meeting of the American Medical Association, Atlantic City, June 7, 1955.

Poliomyelitis,"[3] and "The Danger of Unrecognized Anoxia in Laryngology."[4]

It gives me great pleasure to introduce Dr. Galloway.

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Progress in Otolaryngology

THOMAS C. GALLOWAY, M.D., Evanston, Ill.

It is a great satisfaction to have been singled out for the recognition you have given me today. It is especially so because it comes from men who have had the same aspirations, who have been doing the same kind of thinking and have been striving toward the same end as myself. Such efforts have been based on an accumulating and shared fund of knowledge and are stimulated by the writing, the addresses, the debates, and especially by the personal discussions that develop at these meetings. When any one person honestly contemplates his own part in such widespread activity he should be filled with humility.

Almost no progress in science has been made without the effort, the deliberation, and the investigation of many, except in a limited number of fundamental discoveries. Even Darwin's work in evolution, for instance, was foreshadowed by that of Mendel, Wallace, and a good many others. The field of scientific ideas is like a cultivated land where the harvest may ripen almost all at once but where there must have been a survey and a clearing, a plowing, a cultivation, a fertilization, and much care before the harvester comes. Men may receive ideas, hide them in their subconscious, and later produce them as their own, which indeed they may have become by true assimilation. It is almost an accident who may happen to become the spokesman on a given subject. One may be only a little more articulate or a little bolder than his fellows. Such was the situation when in 1946,[1] with some diffidence, I presented a sort of a potboiler on the subject of the dangers of anoxia in otolaryngology. Rather to my surprise it was well received, because it stated and crystallized thinking that many men had been doing. From the discussion and agreement that arose from this perhaps we have made further progress, as we have with so many presentations before this group.

This seems a fair time to appraise our progress, to ask where otolaryngology is, and how we got here. Some pessimists in our specialty have set a fashion of depreciating its soundness, its achievements, and its future. For two reasons this seems to be bad. In the first place, this discourages those students who might follow in the work in which we have found so much satisfaction, men who would keep our specialty virile, progressive, and accepted for its true place in medicine. In the second place, it is not so that this depreciation is justified. The enthusiasm, the keenness of the discussions we have heard and the papers we have read

Accepted for publication Aug. 17, 1955.

From Northwestern University Medical School and Evanston Hospital.

Read before the Section on Laryngology, Otology and Rhinology at the 104th Annual Meeting of the American Medical Association, Atlantic City, June 7, 1955.

recently, and the forward-looking attitude at this meeting testify that we have not met to liquidate a specialty.

At first blush discoveries of chemotherapy and antibiotics had seemed to deal a serious blow to our work. By great ardor and almost the sweat of blood we had mastered courageous but by no means always successful attacks on serious conditions, such as mastoid complications, otogenic meningitis, and cranial osteomyelitis. But now, with little disappointment, by these new agents we could achieve easier successes.

We had, however, been in danger of becoming mere technicians. The realization of this that came with conquest of certain infections acted like a sort of shock therapy. After a period of preliminary mourning our scientific curiosity again began to assert itself. We took a good look at the unsolved problems in our field, in otology and audiology and speech, in aural and vocal rehabilitation, in malignancy, in allergy, in respiratory complications, in fenestration and functional reconstructive surgery, and many other questions. But most of all we came to realize that we could not loaf in the back waters of medical progress but that we had to strike out strongly in the whole stream of medicine, of which we were a true part. The time had come to make the application of general physiology and the complete body of scientific medicine to our field. But, likewise, we had to realize our responsibility to contribute to the common fund of knowledge. Now not only is the specialty in a most healthy state, in an advancing phase, but as individuals we are in a much more alert state of intellectual activity. We again are trying to become curious, thinking, ambitious, and enthusiastic medical scientists.

In considering medical progress chief emphasis has been given to the product of the laboratory. Beveridge [2] in a provocative small volume has summed up the qualities that lead to success in medical investigation. Among such faculties he lists ability in planning and study, imagination and curiosity, intuition, the power of observation and recognition, and exploitation. Many of us probably at one time had to make a choice between the laboratory and practice. If we chose to work directly with patients I am sure if we progressed we have had to have in some measure most of the qualities that have led to advances in pure research. With such an approach every patient becomes a research problem and every doctor an investigator in living medicine.

Between the two lines of endeavor there are, of course, certain differences. Pure research begins with a plan, usually with a hypothesis, and must have extensive controls. The clinical man has his problem thrust upon him in the sick patient, and circumstances may require him to work under conditions which are far from ideal. Incentives and approaches may differ less than appears superficially. Most scientists would decry a sense of dedication lest they be considered sentimental, but most who succeeded have had it. Although the active practitioner may be motivated only in part by the good he hopes to do, there is no doubt that the stored satisfactions that come from the sufferings eased and the lives saved loom large in his contentment with his calling.

Curiosity must be one of the greatest stimulants. Psychological experiments from the University of Wisconsin indicate that it operates strongly in learning, even at the simian level. Like monkeys in experiments, we keep popping up our heads to see what is going on. But in a better sense our curiosity is Claude Bernard's "torment of the unknown." To the doctor the fear that he knows less than he should or is less skillful or less understanding than he might be is a tremendous driving force. He must constantly learn and investigate and think purposefully in order to render the best service within his power.

The physician must likewise have imagination and intuition if his mind is to take those mental leaps, those short cuts of thought that lead to Webster's "immediate perception of truth without conscious reasoning." Since his is no planned experiment,

he may not too readily apply his rapid conclusions without laboratory control. But in a desperate case he may be so rewarded by unequivocal response that there will be no doubt of the validity of his conclusions.

Chance in the laboratory and in the clinic may present him with rare opportunity. But it will require keen observation to recognize a causal relationship and to exploit it to its fullest.

We have been hearing much about conditioned thinking. The Russians have taken the experiments of their greatest physiologist and by frustration, repetition, denial, and physical degradation so prostituted his methods to channeled mental processes that intelligent men may come to believe that true is false and false true. In a much less deadly sense our own conditioned thinking may lead us into repetitious error. In certain problems a first preconception can block further progress. Answers all too pat which apparently satisfy all doubts may quench imagination and satisfy curiosity so that the mind follows the same preformed paths, often to the same blind ends.

The case of bulbar poliomyelitis was a little like this. Before 1943 laryngologists had not been invited often to consult in this disease. The men who had worked with this condition extensively saw that the vital centers were involved and believed the fault to be chiefly there. On histologic examination of the central nervous system the picture seemed so completely explanatory that further consideration seemed unnecessary. So firm was this idea that even 10 years ago another proposed idea [3] was scarcely given a hearing before medical groups. Wilson [4] in 1932 had suggested that tracheotomy might be useful in this disease, but before laryngologists entered this field very few patients had received such help. So far as one can learn, few laryngologists had even been asked to see such cases.

But conditioned thinking, when it has been correctly applied to one problem, may be adapted to the same situation in other fields. Laryngologists had had considerable experience in respiratory obstruction, developed largely through their care of cases of acute infectious laryngotracheobronchitis. By the studies and efforts of a long list of men they had come to see that the main difficulty here was not infection but respiratory obstruction with anoxia and other secondary effects. From the efforts of Baum, Jackson, Gittens, Orton, Lyman Richards, Davison, Holinger, Neffson,[5] and a host of others the mortality of this disease had been reduced from 30%-50% to nearly zero even before the use of the newer agents. In my own case and that of many laryngologists when confronted with a new problem, bulbar poliomyelitis, the very ignorance of that disease was almost an asset. It allowed us to see this disease as a problem of secretional obstruction. Proceeding on our conditioned experience and treating the disease on that basis, we obtained in many patients such striking relief that we appeared to prove the case almost before we had investigated the alternative. It is true that some men had mentioned the factor of respiratory obstruction in poliomyelitis, but with so little conviction that only a few tracheotomies had been reported.

Laryngologists were then admitted to cooperation in this field, and after the fact that obstruction was so important had been exploited and proved, notably by such reports as that of Priest, Boies, and Goltz,[6] Miller, Bower, and others, such care became an essential part of the treatment. Now many thousands of tracheotomies are done each year, with the saving of many more lives. It was then easy, as Echols [7] has pointed out, to extend these ideas largely developed in the treatment of bulbar poliomyelitis to many other emergency situations. Among those in which respiratory obstruction has come to be recognized as of critical importance are flame injuries, craniocerebral accident, crushed chest, anesthetic misadventures, tetanus, botulism, and other conditions where clear airways are so essential.

After reviewing the present state of our specialty it seems time to abandon any atti-

tude of apology. We should assess our own satisfactions with our calling and point out that it is essential to medicine in general and that it has a right to full consideration. Especially have we a right to ask teachers of medicine to cooperate with us to make students and recent graduates appreciate the opportunities to be broad-gauge medical scientists in the practice of otolaryngology. They should receive a better introduction to our specialty than that they be merely supplied with a flashlight with the implication that they may thus see all that is important in the ears, nose, and throat. We surely may ask that the curious student will early in his training be allowed to develop a facility with proper illumination and instrumentation and visualization that will allow him intriguing glimpses into the important regions of our specialty. He thus will have a real basis for further exploration. And it seems to me we should feel ready to point out to such young men and women that never before in the specialty have the opportunities and the challenges been so great.

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Deafness Following Endemic Parotitis

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La surdité est plus fréquente qu'elle ne devrait être. C'est une infirmité souvent évitable.—PARREL.

In spite of its rarity, toxic infectious involvement of the acoustic nerve should not be considered as of secondary importance, because of the severity it sometimes shows.

Disturbances of audition and equilibrium, deafness, and deaf-mutism after that condition are cited by authors as very important complications of the central nervous system.

Hintze, in 1802, was the first to notice this type of deafness; his patient was a 50-year-old man who had had endemic parotitis. In his book on diseases of the ear, Toynbee was the first to write on the complication. He described the disease precisely but did not explain its mechanism. Toynbee stated, "The special poison that produces the disease generally known as endemic parotitis is very often the cause of complete deafness." Undoubtedly, the nervous system is affected in these cases. Deafness is usually unilateral, sudden, and complete. As a rule, no pathological changes are found either in the acoustic nerve or in the tympanic membrane and cavity.

Mayer wrote that deafness as a complication of endemic parotitis is more frequent than we think, and he cited Bezold's statement that 1.7% of deafness is due to this disease. According to Hubbard, there are 50,000 deaf persons in the United States, in 1000 to 2000 of whom deafness was caused by endemic parotitis; thus, this disease is an important etiological factor in an average of 3% to 5% of cases of deafness. Statistics show that deaf-mutism is more frequent in males; there is no explanation for this incidence.

Voss pointed out the fact that this complication is sometimes unnoticed because the deafness is very often unilateral, with a short evolution period and indefinite otoscopic findings, and many times the patient's difficulty is attributed to his inattention. Undoubtedly, syphilis and infectious diseases are the most frequent causes of deafness and deaf-mutism.

In one such case observed by my colleagues and me, the patient (an 8-year-old child seen on July 7, 1938, at Gaffrée Guinle Hospital) had a strongly positive Wassermann reaction. His pathological history made us believe that endemic parotitis was the exclusive cause; we thought the case remarkable because Heraldo Maciel observed that the Wassermann reaction was predominantly positive in patients with orchitis as a complication of endemic parotitis. It is possible that the same phenomenon may be observed in patients with complications of the ear.

The etiopathogenesis is the same as that of the other infectious diseases.

Endemic parotitis is due to a filtrable virus which is found in the sputum of patients with the disease, as demonstrated by Wollstein in 1916-1918. Philibert described it as a cytotropic and neurotropic virus.

Actually, this disease is believed to be followed, in 65% of cases, by lymphocytic infiltration of the meninges.

There are many reported cases of meningeal reaction during endemic parotitis. One interesting case was related by David de Sanson. His patient showed a meningeal

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reaction but no apparent symptoms of parotitis. De Sanson thought the reaction was a complication of endemic parotitis on the basis of the patient's history of having been in contact with an epidemic environment.

Collet's observation of edema of the glottis after endemic parotitis was useful in the pathological study of this disease.

It has definitely been proved that postparotitic deafness is caused by toxic encephalitis and neuritis. The acoustic nerve presents lesions of neuritis and perineuritis (cochlear and vestibular), and sometimes there are lesions of the facial nerve and of the nerves that pass through the internal auditory tract. These lesions may involve the entire labyrinth or only a part of it.

In addition to paralysis of the facial and cochlear nerves, other paralyses may occur. Liegois noted a case of facial paralysis accompanied by paralysis of the leg on one side and of the arm on the opposite side. He followed this case until cure was achieved.

In acquired deaf-mutism, destruction of the membranous labyrinth and of the sensory epithelium and acoustic nerve endings is generally observed.

Rumbelli was of the opinion that deafness caused by meningitis is due to labyrinthitis without otitis media, resulting in sclerosis of the labyrinth. He had the opportunity to study the petrous portion of the temporal bones of two deaf-mute patients who died 30 and 40 years after they had had scarlet fever. The middle ears of both patients were intact, but the internal ears showed fibrous healing, which induced him to suspect ancient labyrinthitis. At first, it was thought that the acoustic centers of the cerebral cortex were involved by an inflammatory process, as is frequently the case with the testes and ovaries. The actual cause was found to be meningoradiculitis.

Urbantischtsch tried to explain the deafness caused by this disease of the parotid glands as due to a reflex originating from irritation of the sensitive fibers of the trigeminal nerve within the parotid glands. However, this explanation is not reasonable, for it is known that deafness due to endemic parotitis may be observed in patients with apparently normal parotid glands.

Some authors (Voss and others) accorded little probability to the existence of serous meningitis as an intermediary state between the systemic disease and the complication of the ears. (In serous meningitis lumbar puncture shows high spinal fluid pressure and a great number of lymphocytes.)

Briefly, the best explanation seems to be the occurrence of a toxic neuritis of the acoustic nerve, though this has not yet been proved clearly. It is beyond any doubt that the neurotropic action of the virus causes these conditions. This opinion is confirmed by the known existence of many diseases due to virus that may result in deafness. In such cases, the patient begins feeling buzzing and giddiness, and deafness appears suddenly three or four days after the onset of endemic parotitis, coinciding with the relief of the affected gland or with the involvement of the sound one. This complication (deafness) may occur at any stage of the infectious disease, on the side of the affected parotid gland or on the opposite side.

As mentioned above, in addition to complication of the vestibular nerve, one may observe participation of the cochlear nerve as revealed by vomiting, giddiness, and nystagmus, symptoms that may lead to the gross error, as noted by Voss, of suspecting the presence of gastrointestinal diseases.

The lesion is usually unilateral, and in regard to auditory acuity, Castex was of the opinion that cranial perception is the first to be affected. It is very difficult to submit these patients to functional examinations, since they are generally infants.

Treatment with pilocarpine and iodine does not offer good results. The most advisable procedure is lumbar puncture, for it is harmless and a diagnostic as well a therapeutic measure. As Schoental observed, early puncture is a recommended means of avoiding deafness. Clinical treatment must not be abandoned, and it is well to begin training in

lipreading, because we have often observed irreversibility of the lesions.

The prognosis is dark, but in a few cases there is a natural tendency toward cure. The small percentage of observed cases contributes to the lack of knowledge of the condition. Diseases caused by virus have been studied by many researchers, and their conclusions will probably throw some light on this condition, especially on its treatment. We are not able to solve such a difficult problem; however, we call the attention of specialists to this grave complication of the benign disease, endemic parotitis. We have observed this complication in six cases, but we could not at the same time demonstrate the presence of meningitis by cytologic methods.

Happily, in only a few cases does such a serious complication occur, because this disease usually has a benign course.

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Functional Goals in Mobilization of the Stapes

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and
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Since the new technique of stapes mobilization was introduced, in 1952,* one of us (S. R.) has performed over 700 of these operations. On the basis of this experience it can be stated that the degree of hearing improvement attainable by this technique is higher than that attainable by other surgical procedures.

Davis and Walsh [5] and McConnell and Carhart [6] have shown that the most successful fenestration operation still leaves the patient with a residual hearing loss greater by 20 to 25 db. than the cochlear potential (as measured by bone conduction) because of the interruption of the ossicular chain and other structural changes. The "social adequacy" level of 30 db. residual loss following fenestration has been the standard accepted as a successful result. Shambaugh [7] stated that a patient with a postoperative fenestration residual loss of 30 db. is "completely rehabilitated." However, others have long believed that a loss of 30 db. marked the beginning of a handicap in hearing for speech.

During and after World War II, the Armed Forces and the Veterans Administration considered a loss of 30 db. in the better ear sufficient to refer a patient for rehabilitation at a special aural rehabilitation center. Children with an irreversible loss of 30 db. in the better ear are often given lipreading instruction in the schools. It seems, therefore, that surgical restoration of hearing must aim higher than the 30 db. level. The purpose of this paper is to indicate that with mobilization it is possible, by closing the air-borne gap, to achieve higher goals in restoring hearing in patients with all degrees of deafness due to otosclerosis.

GOALS OF THE MOBILIZATION OPERATION

1. To Attain Normal Hearing

Normal hearing is usually accepted as hearing within 10 db. of zero loss for pure tones and speech. In a recent report of results by us,[4] 7 patients out of 44, or 16%, whose preoperative bone-conduction loss did not exceed 20 db. achieved normal hearing following mobilization of the fixed stapedial footplate. In the mobilization operation there is no significant alteration or reconstruction of the anatomical structures of the middle ear. The ossicular chain remains intact, thus permitting the complete restoration of the normal function. Other surgeons † here and abroad have also reported achieving normal hearing after mobilizing the fixed stapedial footplate as described by one of us (S. R.) ‡

The following case operated upon by S. R. is an example of the restoration of normal hearing following successful mobilization:

Case 1.—A 24-year-old woman had bilateral progressive deafness and intermittent tinnitus for 10 years. She heard better in the subway. Her mother was hard of hearing for many years. The patient's average loss for pure tones preoperatively was 38 db. in the right ear, 42 db. in the left. Her speech reception threshold was 45 db. in each ear.

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† Personal communications to the authors.

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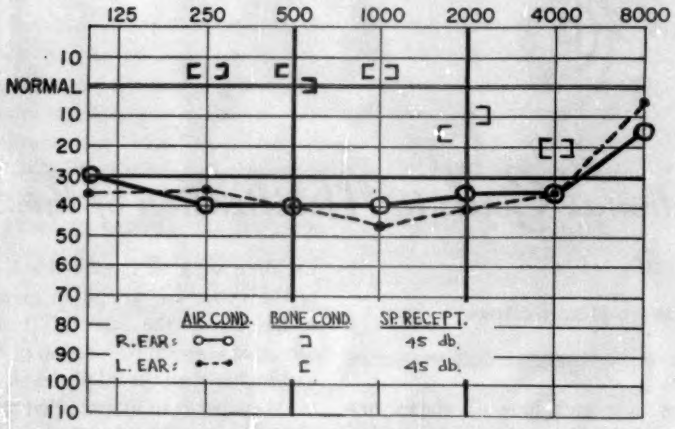

Fig. 1*A* (Case 1).—Premobilization audiogram.

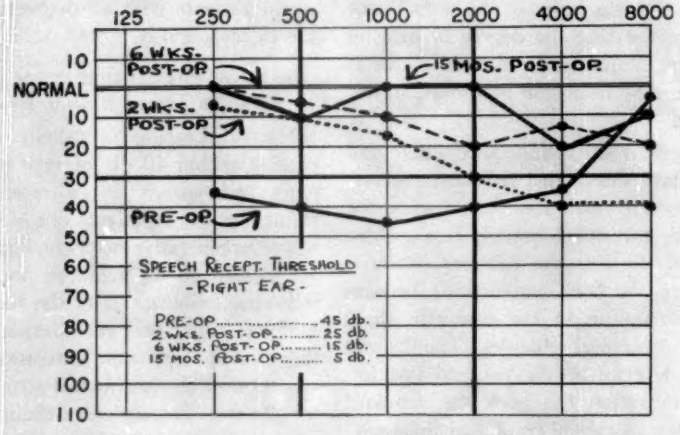

Fig. 1*B* (Case 1).—Postoperative recovery of hearing.

The left rigid stapes was mobilized on April 1, 1954. The hearing improved on the operating table and continued to improve gradually up to a 5 db. loss, a level which has been maintained for 15 months to date. (Figs. 1*A* and 1*B*.)

The second case in this category illustrates that a patient with a bilateral 30 db. hearing loss due to stapedial fixation is sufficiently handicapped to seek relief for her deafness.

Case 2.—A 38-year-old woman, mother of two children, had bilateral progressive deafness for 10 to 12 years and intermittent tinnitus. Her father and sister were hard of hearing for many years. Her preoperative hearing loss for pure tones averaged 28 db. in the right ear and 30 db. in the left ear, with a speech reception threshold of 30 db. bilaterally. At operation May 17, 1955, the left stapes was found to be rigid and was mobilized.

It is interesting to note that although there was a 10- to 12-year handicapping deafness the hearing loss was only 30 db. A patient with such a slight loss is unsuitable for fenestration. Mobilization of the fixed stapedial footplate provided this patient with normal hearing in the ear operated on. (Figs. 2*A* and 2*B*.)

2. To Restore Hearing to an 11 to 20 Db. Level in Patients with Some Evidence of Perceptive Loss in Whom Normal Hearing Is Not Possible

Otosclerotics with a hearing loss between 11 and 20 db. by pure-tone and speech audiometry rarely experience any hearing difficulties. In Cases 3 and 4, whose audiograms are shown, the above level of hearing (11 to

20 db. loss) was realized through mobilization of the stapes despite significant cochlear involvement. The inner ear component of the deafness precludes the attainment of normal hearing, yet mobilization of the fixed stapedial footplate in both cases improved the hearing for speech to as close to normal as was possible (15 and 10 db., respectively).

CASE 3.—A 53-year-old man had bilateral progressive deafness for 10 years. He wore a hearing aid constantly for six years. His average loss for pure tones was 52 db. in the right ear and 50 db. in the left ear. His speech reception threshold was 50 db. in both ears. On Jan. 11, 1954, the very rigid left stapes was mobilized. The hearing has remained at the level shown ever since. (Figs. 3*A* and 3*B*).

CASE 4.—A 39-year-old man had progressive bilateral deafness for 18 years. He wore a hearing aid continuously for 10 years. His preoperative average loss for pure tones was 48 db. for the right ear and 43 db. in the left ear. His speech reception threshold was as follows: right ear, 50 db; left ear, 45 db. On Oct. 16, 1953, the right rigid stapes was mobilized. The hearing has remained at the level shown ever since. (Figs. 4*A* and 4*B*.)

3. To Restore Useful Binaural Hearing

Patients with unilateral deafness, where the hearing in one ear is normal or shows only a slight loss, often complain of difficulty in hearing speech in a noisy environment. This is true also for patients who wear hearing aids, since they too are hearing unilaterally

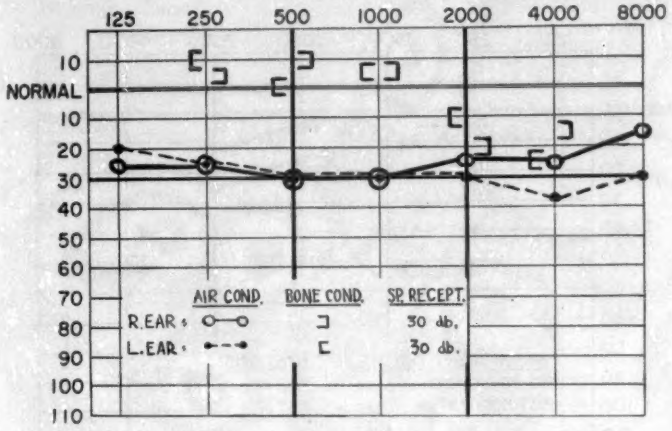

Fig. 2*A* (Case 2).—Premobilization audiogram.

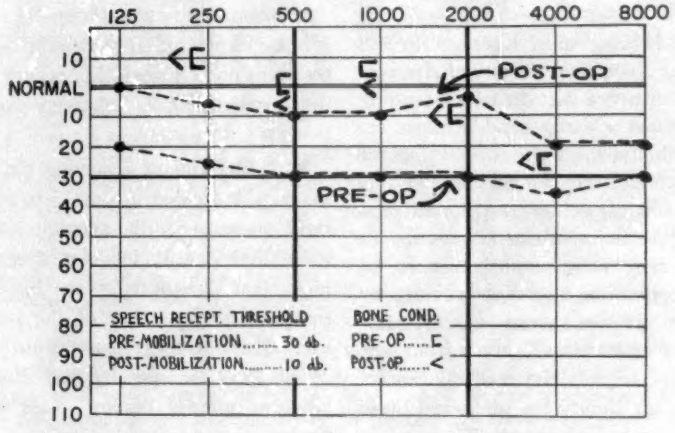

Fig. 2*B* (Case 2).—Postmobilization audiogram.

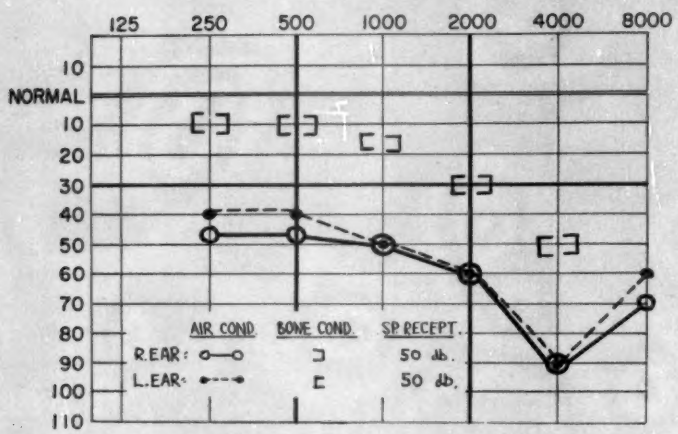

Fig. 3*A* (Case 3).—Preoperative audiogram.

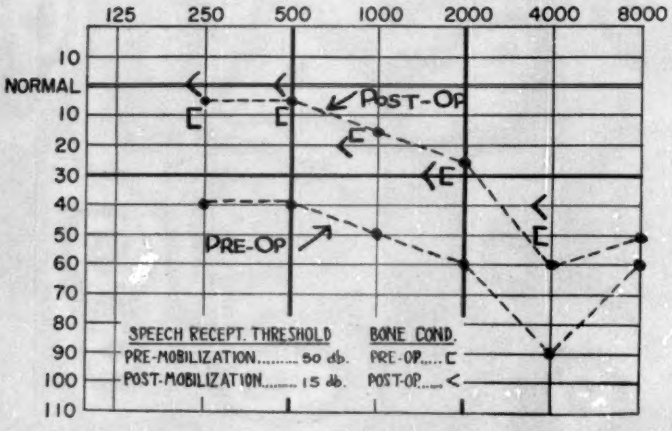

Fig. 3*B* (Case 3).—Audiogram of left ear 17 months after mobilization.

through the hearing aid. Koenig,[8] in the *Journal of the Acoustical Society of America* in 1950, demonstrated that the understanding of speech against a background of noise requires the ability to locate the source of competing sounds. He concluded that this permits the cortical centers to separate the speech from the noise. Such localization in the human can be accomplished only by binaural reception, such as that provided by the two ears hearing about equally. Localization and separation of speech from surrounding noise permits the cerebral centers to suppress with facility the undesired noise in favor of the desired speech. Thus, while it is well known that otosclerotics often hear better in noisy places, this occurs only when the hearing loss is relatively equal in the two ears.

With the aim of achieving binaural hearing it was decided in Case 5 to mobilize the right stapes (52 db. average loss by pure tones and 55 db. loss for speech) in the hope that the hearing in this ear would reach or surpass that of the better (left) ear (28 db. average loss by pure tones and 30 db. loss for speech) and thus result in binaural unaided hearing. This was accomplished successfully.

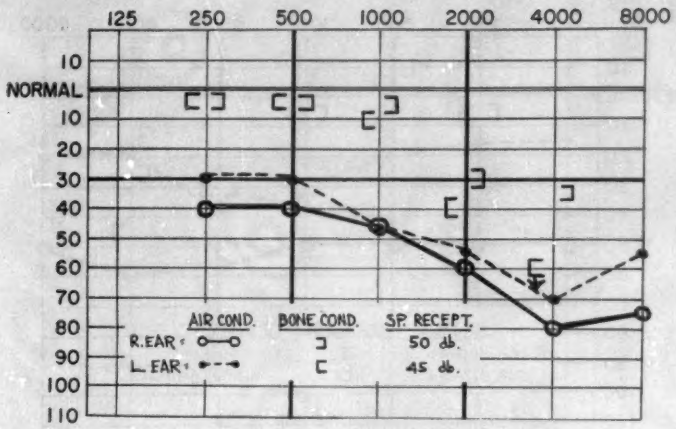

Fig. 4*A* (Case 4).—Premobilization audiogram.

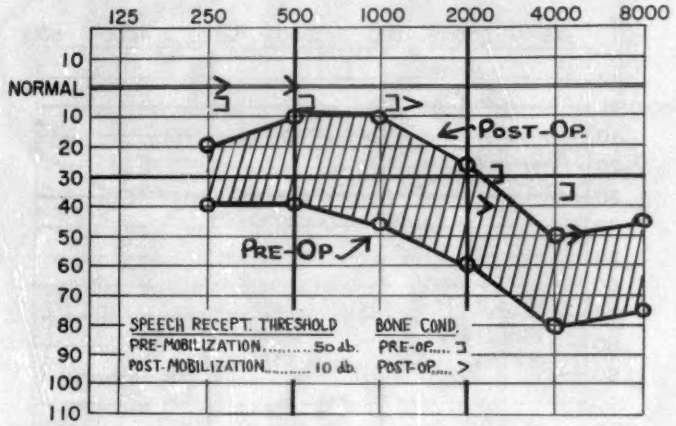

Fig. 4*B* (Case 4).—Audiogram of right ear 13 months after mobilization.

Case 5.—A 51-year-old woman with a history of deafness in the right ear for 14 years and in the left for 1 year. On Sept. 20, 1954, the right rigid stapes was mobilized and the hearing raised to the level of the better (left) ear. (Figs. 5*A* and 5*B*.)

Case 6.—A 42-year-old woman had bilateral progressive deafness for 25 years. A successful left fenestration by another surgeon was performed in 1945. Nine years later (1954) she decided to have the mobilization operation on the right ear. The speech reception threshold of the left (fenestrated) ear was 30 db., with an average pure-tone loss of 30 db. The right ear showed an average pure-tone loss of 50 db. and a speech reception threshold of 55 db. Successful mobilization of the right stapes on Dec. 3, 1954, resulted in hearing in the right ear of 25 db., which was slightly better than the hearing in the successfully fenestrated left ear. Binaural hearing was thus achieved. (Figs. 6*A* and 6*B*.)

4. To Remove the Conductive Component (Stapedial) in Mixed Deafness (Cochlear and Stapedial)

Case 7 best illustrates a severe mixed deafness where the hearing loss due to stapedial fixation was completely eliminated by mobilization, leaving the cochlear deafness. The improved hearing following elimination of that portion of the deafness due to stapedial fixation allowed more effective use of the hearing aid.

Case 7.—A 28-year-old man had bilateral progressive deafness for over 10 years. An unsuccessful right fenestration performed in Bucharest in 1945 was followed by facial palsy, which recovered in six months. There was severe tinnitus in both ears. On Aug. 10, 1952, the left stapes was mo-

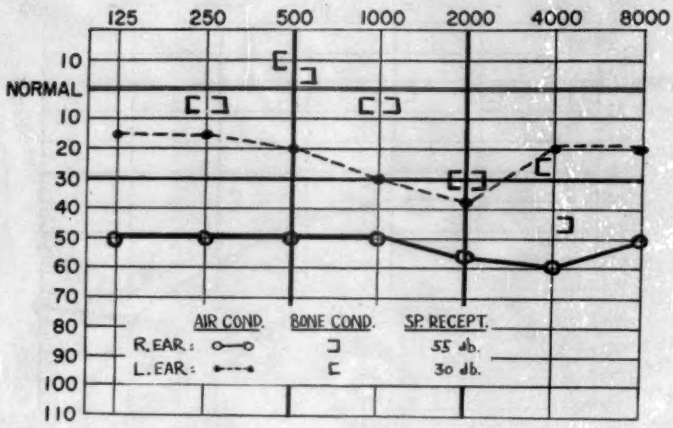

Fig. 5*A* (Case 5).—Premobilization audiogram.

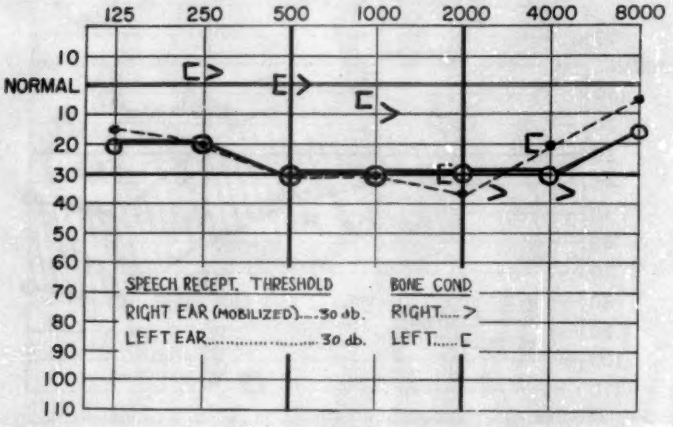

Fig. 5*B* (Case 5).—Audiogram six months after mobilization.

bilized. Hearing dramatically improved on the operating table. The hearing by air conduction in the left (unfenestrated) ear preoperatively showed an average pure-tone loss of 82 db. Hearing by bone conduction, though poor, indicated the possibility of improving his hearing to the degree that the conductive element might be removed. The hearing by air conduction postoperatively showed an average loss of 47 db. for 500 through 2000 cps, with hearing at 20 to 25 db. at 125 and 250 cps, respectively. The improvement is significant in terms of the value to be derived from the use of a hearing aid. (Fig. 7.)

5. To Permit Successful Use of Hearing Aid in Extreme Deafness

The gain or amplification of the strongest commercial hearing aid is insufficient to provide comfortable hearing for ordinary conversation in a patient with a hearing loss in the better ear greater than 85 db. Any significant improvement in hearing in such profound deafness increases greatly the benefits to be derived from the use of a hearing aid.

Case 8.—A 45-year-old woman had bilateral rapidly progressive deafness since the age of 18. The speech reception threshold was 100 db. in the left ear and 95 db. in the right. The speech discrimination test had to be performed at only 10 and 15 db. above the speech reception threshold of the right and left ears, respectively. At these low sensation levels her discrimination scores were relatively high (58% and 62%), suggesting the feasibility of mobilization. The patient complained of

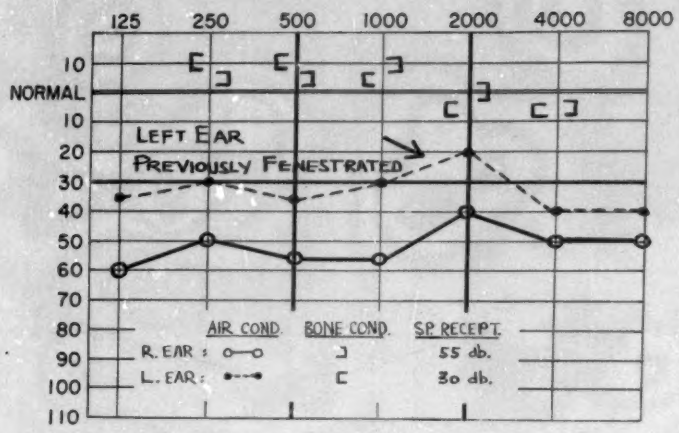

Fig. 6*A* (Case 6).—Premobilization audiogram.

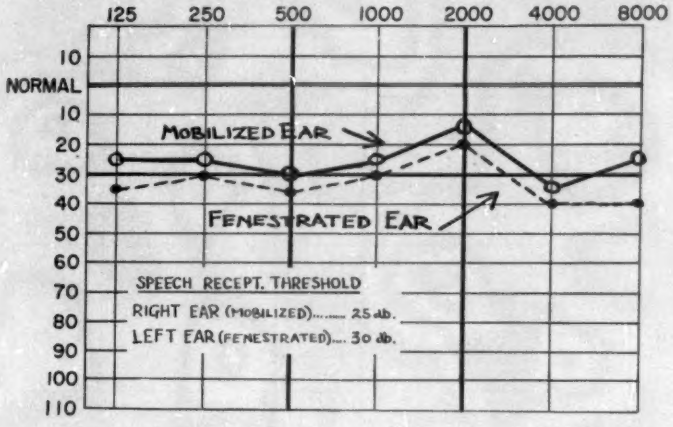

Fig. 6*B* (Case 6).—Audiogram five months after mobilization.

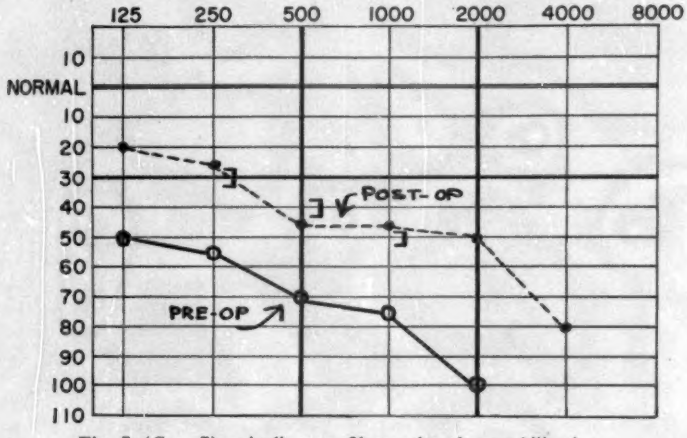

Fig. 7 (Case 7).—Audiogram 21 months after mobilization.

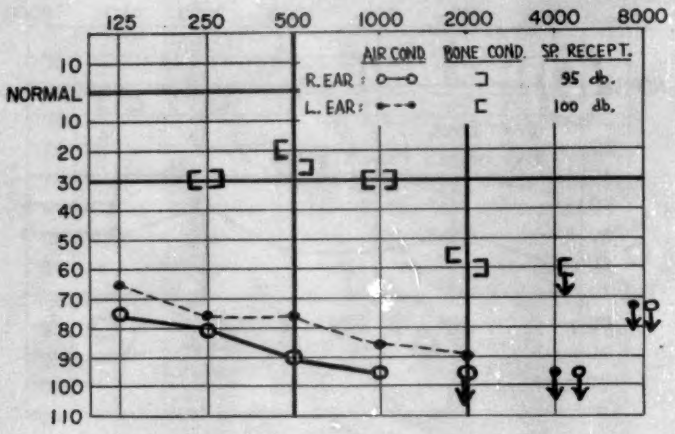

Fig. 8*A* (Case 8).—Premobilization audiogram.

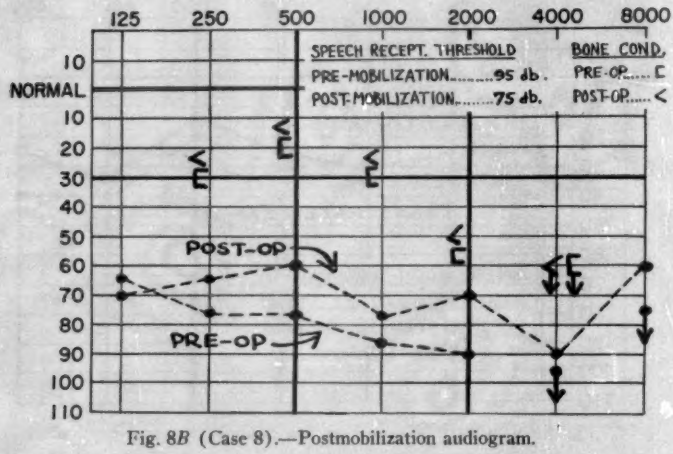

Fig. 8*B* (Case 8).—Postmobilization audiogram.

her inability to hear adequately with maximum amplification of her hearing aid.

On Nov. 10, 1954, her very rigid left stapes was mobilized. The pure-tone average in the ear operated on rose from 83 to 68 db. loss, and the speech reception threshold rose from 95 to 75 db. The discrimination score in the ear operated on rose from 62% to 94% at the same testing level used preoperatively: 110 db. above zero hearing loss for speech. Since mobilization the patient reports greatly improved hearing with the hearing aid. (Figs. 8*A* and 8*B*.)

The following case demonstrates the need for rehabilitation follow-up when a profound deafness is partially relieved by surgery.

Case 9.—A 34-year-old woman had an unsuccessful fenestration performed by another surgeon in the right ear in 1943, with an unsuccessful revision of the fenestration in 1948. The patient reported that her hearing receded considerably following the revision. The decision to attempt stapes mobilization on the left ear was based upon the relatively favorable bone conduction and the excellent speech discrimination score of 88% obtained at only 20 db. above her threshold for speech on that ear (110 db. above normal threshold for speech, the limit of the testing equipment).

On Aug. 4, 1954, her left stapes was found extremely rigid and was mobilized. Two months later the patient reported that she could now hear "muffled voices." The hearing in the left ear showed improvement both by pure tones and speech audiometry. The hearing by pure tones improved from a preoperative average of 77 db. to a postoperative average of 60 db., while the speech reception threshold of 90 db. became 60 db. postoperatively.

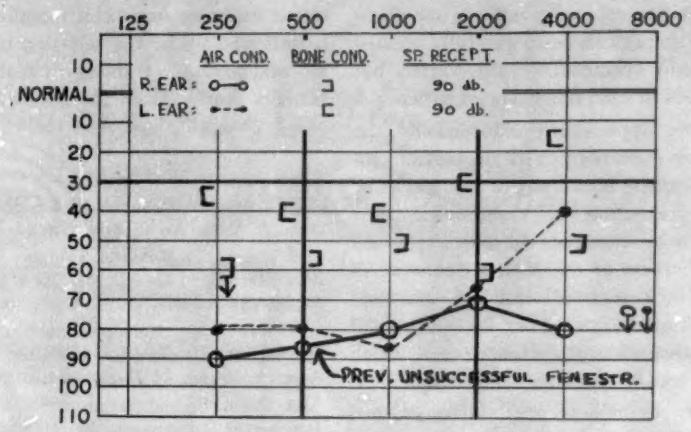

Fig. 9*A* (Case 9).—Premobilization audiogram.

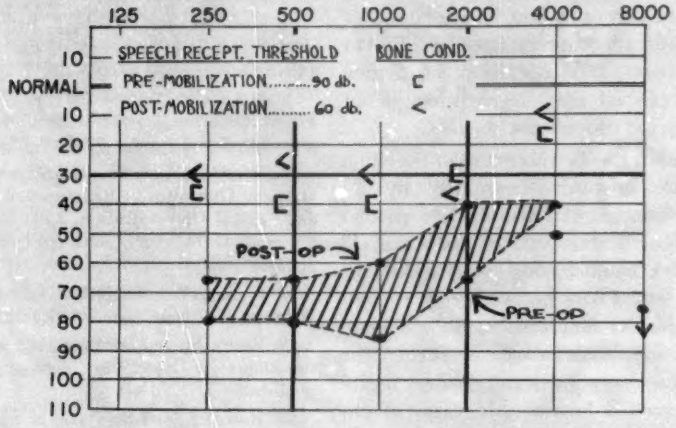

Fig. 9*B* (Case 9).—Audiogram 10 months after mobilization.

Before the mobilization operation the patient had not been able to hear enough with a hearing aid to warrant its use and had given it up. After the mobilization of stapes she again tried to get along without a hearing aid but found little noticeable improvement in her unaided hearing. When she was tested with a hearing aid, however, she found no difficulty in hearing and was able to participate fully in conversations. The selection of a suitable hearing aid following the moderate improvement in hearing by mobilization of the fixed stapes in patients with such profound deafness is an essential part of the otological follow-up treatment. (Figs. 9*A* and 9*B*.)

SUMMARY

The ultimate goal of any treatment for otosclerotic deafness is the restoration of normal hearing in both ears. Mobilization of the fixed stapes is capable of achieving this goal where there is as yet no cochlear (perceptive) involvement. The goals of mobilization vary with each patient's hearing potential. Through successful mobilization it is possible to realize the full hearing potential in the various degrees of otosclerotic deafness.

The selection of cases for surgery and the results to be expected must be based upon the most careful and complete audiological investigations. This implies testing under stabilized conditions with low ambient noise, with calibrated pure-tone and speech audiometry, and by trained personnel, preferably someone other than the surgeon. With such

quantitative control now available, mobilization of the stapes can be successfully applied to the broad spectrum of the varied but unique needs of each handicapped patient.

The wide applicability afforded by the mobilization operation for all degrees of otosclerotic deafness broadens our horizon. The increasing attention to mobilization now being given by otologists all over the world since publication of the Rosen technique in 1952 and subsequent clinical and statistical reports § should improve our knowledge and skill to realize these goals more often, which may then lead to even higher goals.

In order to achieve the aforementioned goals, a high level of skill in performing this operation must first be attained. Mobilization is not an easy operation. There can be no substitute for adequate cadaver practice to learn the mobilization operation, no matter how accomplished the surgeon may be in other otological procedures.

Mobilization can be successful in the same types of cases in which fenestration can succeed. Mobilization can also restore normal hearing. Mobilization can be successful in cases with a handicapping hearing loss of 30 db. or less, a loss not yet great enough to be suitable for fenestration. In moderate and profound otosclerotic deafness, the mobilization operation can restore useful aided or unaided hearing. In cases of extreme deafness successful mobilization must be followed up by the selection of a hearing aid and auditory training to realize the full benefits gained from the operation.

101 E. 73rd St. (21).

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The Reaction of the Cochlea Following a Fenestration Operation

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The most important contributions to the present surgical treatment of otosclerosis have been made by Holmgren, Sourdille, and Lempert. Their pioneer work has led to the development of an operation which today is being performed throughout the world. If a fenestration operation is performed successfully, the resulting improvement of hearing causes a total change in the patient's everyday life. This operation, therefore, in its present form is of paramount importance for hard of hearing otosclerosis patients.

The operative results obtained are highly variable. The literature shows that a satisfactory improvement of hearing is obtained in 50%-85% of the surgical cases. The considerable variation is attributable partly to differences in the standards used in the interpretation of results.

During the period April, 1947, to April, 1952, I performed 402 fenestration operations. The results obtained satisfied the patients in 76.4% of cases. However, it is not my intention to discuss the favorable aspects of the operation in this survey; it is the unfavorable aspects with which I am here concerned.

All surgeons who perform fenestration operations are aware of the great difference between the patient with improved hearing and the unfortunate one who has to put up with unfavorable operative results. In the unfavorable cases the conduction deafness is, as a rule, transformed into perceptive deafness, which is often incompatible with the use of a hearing aid. The psychic effect of an unsatisfactory outcome, however, is at least as important as the resulting condition of the auditory organ. The tremendous disappointment with which the patient has to cope following failure of a mode of treatment on which his hope had been placed should not be underrated. It is for this reason that the 23.6% unfavorable operative results are discussed here.

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In my series, results obtained were unsatisfactory in 96 cases. The causes of failure were the following:

(*a*) Osseous closure of the window in seven cases, i. e., 1.72%. This low percentage indicates that this condition is no longer the main problem in fenestration surgery. I always extend the length of the fistula in the horizontal semicircular canal in the manner introduced by G. Passe. The endochondral bone around the fistula is drilled away to a considerable depth (Shambaugh).

(*b*) Incorrect indication in three cases.

(*c*) Damage to the membranous labyrinth in five cases. In no case did this injury give rise to total loss of hearing. In another patient (the sixth) the membranous labyrinth was even torn off completely, in spite of which hearing improved. It has remained satisfactory up to this day, six years after the operation.

(*d*) The postoperative reaction of the labyrinth in 81 cases. In my opinion, this is the most important cause of failure in fenestration operations.

THE POSTOPERATIVE REACTION OF THE LABYRINTH

I am purposely avoiding the term labyrinthitis, which refers to an inflammation. In all cases I ask myself whether a true

inflammation is in fact involved. The designation, postoperative reaction of the labyrinth, is a more neutral one because little is actually known of what happens in the labyrinth, and particularly in the cochlea, after a fenestration operation.

The postoperative reaction of the labyrinth is a generally known phenomenon. Hearing improves immediately after the operation, and improvement continues for a few hours or days, whereupon perception diminishes, particularly for frequencies above 1000 Hz. In the favorable cases hearing is restored after 7-14 days. The deeper the fall and the more prolonged the decreased hearing, however, the less favorable is, as a rule, the final result. Audiometry during this period reveals unmistakable recruitment. This suggests an anomaly of the hair cells in the cochlea. This anomaly of the internal ear did not exist prior to the operation and must, therefore, be a result of the opening of the labyrinth. The only direct communication between the field of operation and the cochlea is via the perilymph. This fluid surrounds the membranous labyrinth of the vestibular organ and the cochlea. It is, therefore, most likely that changes in the composition of the perilymph give rise to the ensuing perceptive deafness

Shambaugh was among the first to study this problem. He presumed that blood had entered the fistula and formed deposits on the Reissner membrane. An argument against this theory is found in the fact that blood on the Reissner membrane need not immediately cause degeneration of the hair cells; yet recruitment proves to be present from the beginning. Another theory advanced is that of the entrance of histamine or histamine-like substances from the flap, with resulting cochlear damage. This conception of the postoperative reaction of the labyrinth led several surgeons, e. g., Wullstein and Antoli-Candela, to cover the fistula with a free flap rather than a pedicled flap. I have seen no improvement resulting from this technique.

Since changes in the composition of the perilymph are most probably the cause of the postoperative cochlear reaction, it is of importance to investigate the composition of the perilymph and the site of its production.

There are considerable discrepancies in the literature regarding the origin of endolymph and perilymph. Arnvig [1] and Dodds [2] contend that perilymph is a product of the cerebrospinal fluid. Waltner,[3] who examined human perilymph obtained during Day's operation, stated that perilymph is only partly a product of cerebrospinal fluid, i. e., only so far as the crystalloids are concerned. The source of the proteins is obscure, because the level is twice as high as that of cerebrospinal fluid. Mygind [4] and Graf [5] maintain that perilymph is secreted in the spiral ligament.

Ledoux [6] and Paludetti [7] assume that the stria vascularis is the source of the endolymph, whereas Dodds [2] and Mygind [4] are of the opinion that the endolymph is a product of the membranous labyrinth. Saxén [8] reached the conclusion that endolymph partially results from diffusion of perilymph through the Reissner membrane. There is a difference in chemical composition however; it is, therefore, contended that a part is secreted by the cells of the organ of Corti. Saxén maintains that no proof has ever been produced to demonstrate that the stria vascularis is the source of the endolymph. Waltner,[9] on examining cats, found secretion of endolymph not only in the stria vascularis but also in the maculae of the sacculus and the ventriculus, in the cristae of the semicircular canals, and, especially, in the first convolution of the cochlea, near the round window.

Aldred, Hallpike, and Ledoux [10] performed punctures on cats and found an osmotic pressure (expressed in grams of NaCl per 100 ml. of water) of 0.994 for blood, 1.017 for cerebrospinal fluid, 1.046 for perilymph, and 1.058 for endolymph. Dodds [2] obtained the same values and found the ratio of ion concentration between endolymph and perilymph to be Na 1 : 10, Cl 9 : 10, and K 25 : 1. This high K concentration is not seen in any fluid elsewhere in the body; it resembles that of the intracellular fluids. Waltner [9] found that the total N concentration of the peri-

lymph in cats and dogs is twice as high as that of cerebrospinal fluid. Smith, Lowry, and Wu [11] investigated the composition of perilymph and endolymph in cats. The K concentration of the endolymph was 30 times as high as that of the perilymph. The Na concentration, however, was only one-tenth that of the perilymph or cerebrospinal fluid. Perilymph and cerebrospinal fluid had the same K, Na, and Cl concentrations.

It is a generally accepted custom to keep the wound cavity clean during the fenestration operation by lavage and suction drainage with isotonic NaCl solution (0.9 gm/100 ml. of water). This fluid is also used when the fistula is open. The above has made it clear that isotonic NaCl solution is considerably different from perilymph as regards composition and osmotic pressure. As a result of lavage and suction drainage while the labyrinth is open, part of the perilymph will be replaced by the NaCl solution. The composition of the perilymph will be altered to an increasing extent with more frequent lavage. One should, therefore, refrain from use of constant lavage and such drainage, in my opinion, as long as the fistula is open. The less lavage the better. The change in the composition of the perilymph may be compared with the experiments of Walzl,[13] who washed the perilymph space in cats with a hypotonic saline solution and was able to demonstrate a decrease in the cochlear potentials.

The effect of partial replacement of perilymph by a 0.9% NaCl solution on the composition of the endolymph is unknown. It is likely, however, that the composition is altered. Various investigators maintain that the membranous labyrinth is permeable to crystalloids and, according to some, also to proteins. It may well be that this has serious consequences for the hair cells in the cochlea, especially when the source of perilymph and endolymph is unable to restore quickly the original composition. This may offer an explanation for the cochlear degeneration following fenestration operations.

On the basis of these considerations, the lavage fluid by open fistula was modified in such a manner as to approach the composition and osmotic pressure of perilymph. A. Ringer-Locke solution was selected for this purpose; 1.5 gm. of NaCl and 0.2% serum albumins were added per liter.

As far as I know, only Lamp [12] has paid attention to the composition of the washing fluid. He used a solution of NaCl and sucrose which, at 37 C, is isotonic with perilymph.

Since April, 1952, I have performed 77 fenestration operations using this fluid at the moment the fistula was opened. The results were satisfactory in 71 cases and unsatisfactory in 6 cases.

The cause of the less satisfactory result in these six cases was studied. Two patients initially showed satisfactory improvement of hearing. In one, this improvement was partly lost owing to otitis media a few months after the operation. The other patient developed internal ear deafness (bilateral) within three months. The cause has remained obscure. The other four patients had combined middle ear and inner ear deafness, and it was clear, prior to the operation, that the chance of success was but slight.

The most important point, however, is found in the fact that failure of the operation was in no case attributable to a postoperative reaction of the labyrinth.

The average functional gain on the 10th postoperative day following the use of this fluid was 9.3 db. higher than in cases treated with the old isotonic saline solution. This indicates a considerably less severe postoperative reaction.

The number of cases may be somewhat small for reaching definite conclusion, but the results are at any rate encouraging, particularly, when it is borne in mind that in the first series unsatisfactory results were attributable in 20% of cases to the postoperative reaction of the labyrinth.

The impression was gained, moreover, that postoperative disturbances in equilibrium have been less serious since use of the modified lavage fluid was introduced.

SUMMARY

During a five-year period, 402 fenestration operations were performed. Results were

unsatisfactory in 23.6% of cases. The main cause of failure was the postoperative reaction of the labyrinth, which is probably attributable to lavage when the horizontal semicircular canal is opened with a fluid differing in composition from perilymph and, moreover, hypotonic. A fluid was, therefore, made up which approached perilymph in these properties. Only this fluid was used when the fistula was open. The outcome was that unsatisfactory results among the last 77 fenestrations were in no case attributable to the postoperative reaction of the labyrinth.

Herenweg 92.

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Vertigo

A Further Contribution to Therapy Based upon Its Physiopathological Aspect

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INTRODUCTION

In my three earlier articles,* I reported on the results of an investigation on the physiopathological aspects of postoperative wounds, vertigo associated with peripheral vestibular and peripheral vestibular cochlear disease (Ménière's syndrome) and head and neck pain.

These articles contained a presentation of Revici's † theories and hypothesis on the pathogenesis of pathological foci and showed particularly how this hypothesis could be related to the above problems in otolaryngology.

These theories were presented in sufficient detail to enable the reader to understand the methods employed in the investigation of a pathological process and its manifestations and to relate them to the existence therein of an active abnormal metabolism. The abnormal metabolic process is manifested in two opposite directions. A dualism appears for the symptoms and the processes involved which manifest themselves as a local acidosis or a local alkalosis in the focus.

In the first article, it was shown how *n*-butanol (*n*-butyl alcohol) was used to control the alkaline postoperative pain. In the second and third articles on vertigo and head and neck pain, the beneficial effects of lipids with acid or hydroxyl polar groups, when used to influence the abnormal metabolism of foci that manifest themselves as acid or alkaline symptoms, have confirmed Revici's concepts concerning the place of lipids in the pathogenesis of the above conditions.

These concepts present a new approach to the study of pathological states in which many etiological factors and mechanisms are generally recognized and accepted.

The subject of vertigo is of such compelling interest that I continued the investigation after the publication of my latest article, in 1953. As in my previous article on vertigo, I started with Revici's concept that the abnormal metabolism in the vestibular labyrinth was responsible for the induction of vertigo. By employing the same methods of research and diagnosis of the character of the symptom produced by the pathological focus, I was able to correct the abnormal metabolism in a majority of cases.

In addition, I utilized a group of substances which Revici has recently developed and which belong to the general group of liposides. These substances, which correspond to normal, abnormal, and artificial liposides, have different effects on the abnormal focus. When these substances were employed to influence an appropriate symptom, their action was immediately apparent and again confirmed Revici's ideas of the importance of lipids in the formation of pathological foci and the restoration of the metabolic imbalance in such foci by the appropriate use of these various lipids.

On account of the radical departure from accepted ideas of the pathogenesis of abnormal foci, periodic reports of Revici's methods and their application in the field of otolaryngology are sufficiently interesting to be presented. At this point, I refer the reader to my earlier articles for more detailed

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† References 4 through 8. Revici, E.: The Organization in Nature: Its Relationship to the Abnormal Focus, to be published.

information concerning Revici's hypothesis, methods of research, and the substances utilized to correct the metabolic imbalance of the abnormal focus.

In concluding these introductory remarks, I wish to add that Revici's findings have been confirmed by me in a smaller series of unreported cases of itching, asthma, and hemorrhage associated with pathological foci. Other interesting but unreported observations in the field of otolaryngology have also confirmed Revici's experimental findings at the Institute of Applied Biology, Brooklyn, as well as his application of these findings to disease states in other fields of medicine.

METHOD OF STUDY

When the patient complaining of the symptom of vertigo was examined and the clinical diagnosis of peripheral vestibular disease or peripheral vestibular cochlear disease was made, the next step naturally followed, that is, to determine whether a metabolic imbalance could be recognized and, if so, whether the symptom of vertigo was of an acid or an alkaline character.

In this study, the same approach was utilized as was used in my previous studies on pain, headache, and vertigo. First, the history was evaluated in order to ascertain whether the evolution of the symptom accorded with Revici's theory that many symptoms vary in intensity according to the time of day, the relationship of the intake of food to the increase or decrease of the intensity of the symptom, in this case, vertigo. Active foci of disease, i. e., abscessed teeth or gall-bladder disease, that might bear on the onset of the disease, and basic personality changes which had existed for long periods of time, such as hypochondria, depression, anger, or fear, that could have a bearing on increasing the intensity of the symptom, were sought. This portion of the investigation was helpful. In many cases, this part of the history gave me a clinical clue to the alkaline or acid character of the lesion and was confirmed as the study of the case developed.

The technique utilized in my 1953 series of cases of vertigo, which consisted of a comparison between the simultaneous changes in the intensity of vertigo and hourly changes in the pH of the urine, was not used in the present series. No opportunity was afforded to study a case under hospital conditions. Occasionally, I examined three or four hourly specimens of urine taken when the vertigo was most marked or at a time of day that the vertigo was least marked. If the pH values were acid or alkaline, as the case might be, I tentatively accepted the symptom as alkaline or acid if the symptom was most intense when the specimens of urine were collected.

Most patients cannot judge the variations at hourly intervals in the intensity of the symptom of vertigo. In a few cases satisfactory curves were obtained, and I refer the reader to my earlier article on vertigo in which such curves were reported and analyzed. This method is impractical for routine use in ambulatory patients.

I have continued the use of the same substances as were employed in my earlier study on vertigo to determine the acid or alkaline character of the symptom. Their action was generally rapid and definite, and the metabolic pattern of the symptom could be established by this means. Predominantly hydrosoluble acidifying substances, such as ammonium chloride (NH_4Cl), in 45 to 60 grain (3 to 4 gm.) doses and occasionally alkalinizing substances, such as sodium bicarbonate ($NaHCO_3$), in 60 to 90 gm. doses were used, but the first-named substances were generally preferred.

The various substances utilized to evaluate the symptom of vertigo according to its alkaline or acid character were divided into two groups. The first group of substances consisted of nikethamide (Coramine) and *n*-butyl alcohol. These substances had similar effects on the intensity of pain and vertigo and were reported on in my earlier articles. They increased the intensity of acid pain or vertigo and decreased the intensity of alkaline pain or vertigo. The second group, which showed opposite effects on the above

symptoms, consisted of sodium thiosulfate and sulfur in oil. Thus, use of these two groups of substances, which Revici had shown to have antagonistic effects and which were utilized in my earlier studies, was continued as a diagnostic method of the character of the symptom of vertigo in this study also.

It was almost constantly observed that a patient who responded to substances of one group with an increase or decrease of the symptom of vertigo responded in the opposite manner when the substances of the other group were utilized. The patient who responded with an increase in the intensity of the symptom of vertigo to nikethamide or *n*-butyl alcohol showed a decrease in the intensity of the symptom with sodium thiosulfate or sulfur in oil.

There were occasional cases that did not respond to either of these groups. In this series, such cases were not considered to have a manifest metabolic imbalance in the pathological focus and were discarded. If a patient responded to one group of substances, that patient always responded in an opposite manner to the other group.

In my previous communication on head and neck pain, I reported on the use of dihydroergotamine (D.H.E. 45) and a preparation of methanesulfonate salts of dihydroergocornine, dihydroergocristine, and dihydroergokryptine (Hydergine) and the different actions of each at the clinical level. Dihydroergotamine seemed to decrease the intensity of an alkaline symptom and increase the intensity of an acid symptom. Hydergine had an opposite effect. In some of the cases in this series, these substances were utilized to help establish the acid or alkaline character of the symptom but were not used as part of the definitive treatment.

The foregoing remarks indicate the method of arriving at the diagnosis of the alkaline or acid character of the symptom. The next and logical step was the continuation of the use of the above substances, and of other substances that have recently been developed by Revici as part of the study of this condition, in treatment to control the symptom of vertigo.

These recently developed substances were taken and developed from various sources by Revici and shown by him to correspond in certain aspects to the pathological condition. In my previous paper on head and neck pain, I reported on the use of the nonsaponifiable fraction obtained from natural sources and of the conjugated fatty acids from cod liver oil when as "abnormal" lipids combined with sulfur they represented artificial liposides.

The substances that were recently developed by Revici are tabulated in Table 13. As may be noted from the table, the substances are varied in origin. They have been derived from plants, animal tissue (e. g., pigs and cows), fish (e. g., carp and cod), and commercial sources. Some have been transformed and others have been synthesized by Revici. As Revici demonstrated, they all act in a manner similar to that of the large class of substances which he termed "liposides," and which he classified first according to their polar group and then according to their nonpolar group.

Despite the different chemical structures of the substances, they all influence the local change in the pH at the site of the pathological focus. This action is strongly related to their polar groups. For example, the chemical structure of xanthophyll is widely different from that of *n*-butanol, but the action of both these substances affects the local pH in a like manner; that is, both depress the pH at the site of the pathological focus. Other effects are seen according to the nonpolar group, from the chemical point of view. As I noted earlier, these substances have a greater intensity of effect than the less complex substances I had utilized in my earlier studies.

These recently developed substances were utilized, as were the earlier substances, to help in determining the acid or alkaline character of the symptom of vertigo. In this way, I could quickly estimate the effect of a substance on the symptom intensity and also determine the probability of an ultimate good

result with that substance as a mainstay of the treatment.

The utilization of these substances provided additional data confirming Revici's theories on the role of "abnormal" lipids in the metabolic imbalance that exists in pathological tissue, and also confirmed his studies on the greater intensity of action of the great group of substances that he classified as artificial liposides. I have repeatedly observed the favorable effects of these substances, and their action was very rapid, in a matter of three to five minutes affording relief in the painful syndrome or ameliorating the symptom of vertigo. However, because of their more profound effect upon the lesion, I preferred to use them with the intention to change the lesion itself, reserving the less complex substances for diagnostic purposes.

Use of the Substances in Diagnosis of the Character of the Symptom and Treatment

Use of Substances for the Alkaline Group.—If preliminary investigation indicated that the symptom was alkaline, nikethamide was administered, either 15 to 20 drops by mouth or 1.5 cc. by injection. This substance was used as a preliminary diagnostic test to see how the symptom was affected. If the response was immediately beneficial (after 15 to 20 minutes), the symptom of vertigo was considered to be alkaline in character. In this case, treatment followed with one of the liposides having alcoholic polar groups, e. g., *n*-butyl alcohol, 7% solution in water, 15 to 20 drops four times a day. If the symptom was not controlled, additional doses were administered between those on the regular schedule at intervals of 20 minutes until the symptom was controlled, and then the patient reverted to the regular schedule. This procedure was the mainstay of treatment for the alkaline cases.

When the vertigo was severe and accompanied by vomiting and prostration, 7% *n*-butyl alcohol in saline was used intramuscularly or 1 cc. of dihydroergotamine was tried. The *n*-butyl alcohol in saline was then given, 2 cc. or more every hour or two intramuscularly.

In addition to the oral medication, I administered the injectable nonsaponifiable fractions intramuscularly in 1 cc. doses once or twice weekly.

Use of Substances for the Acid Group.—If the symptom was aggravated by the administration of nikethamide or *n*-butyl alcohol, it was considered to be of acid character. The diagnosis was generally confirmed by the amelioration of the symptom with the administration of substances of the acid group, either sodium thiosulfate or sulfur in oil. I prescribed 15 to 20 drops of a solution of 10% sodium thiosulfate in water, continued four times a day. If recurrence of the symptom required it, supplementary doses were administered at shorter intervals, or subcutaneous injections of a sterile 4% solution of sodium thiosulfate in doses of 1 cc. were given. In addition, a solution of 0.5% sublimed sulfur in vegetable oil (Wesson Oil) was given orally, 2 to 5 drops three times a day, dropped on the tongue or given on a piece of soft bread.

In this series, intramuscular injections of 1 cc. of sulfur in oil were given once or twice a week. The recently developed preparations, such as various sulfur compounds, were used similarly. The use of these liposides facilitated the treatment of acute attacks of vertigo. The beneficial effects were more sustained, and the control was more rapidly attained with their utilization. Table 13 lists all the substances administered in this series.

The source of the material, its form of administration, and the dosage, which was usually 1 cc. of the substance dissolved in oil, are given in Table 13. The oil solutions were put up in sterile ampules. In the table a code number is given to facilitate discussion of the substances and the cases in which they were utilized are also shown.

Treatment

It can be readily concluded from the foregoing discussion on the diagnostic method employed in determining the character of the symptom that definitive treatment of the

lesion would be either a continuation of the administration of the same substances as were utilized in arriving at a diagnosis of its character or the administration of other substances. The ultimate goal was to control the symptom of vertigo in intensity and its tendency to recur, as is often the case. With increasing experience in Revici's method and with the recent substances he has developed, it was hoped that the initial objective of symptom alleviation could be extended to change the course of the evolution of the disease process in the labyrinth. The first part of the objective was confirmed in that the symptom could be controlled in both intensity and duration. The second part of the objective was apparently attained in a fair proportion of the cases, because no recurrences were noted in either the peripheral vestibular or the peripheral vestibular cochlear types of cases during the period of observation.

The same two groups of substances were administered to the patients with vertigo as had been used in arriving at the diagnosis of the character of the symptom. In addition, the recently developed substances, which in Revici's classification are artificial lipoacids or lipoalcohols, were administered by injection. Thus, the patients with alkaline vertigo were treated with lipoalcohols or lipobases, and the patients with an acid type of vertigo were treated with lipoacids.

The result of the treatment was judged by a consideration of two factors: (1) as was noted above, the immediate effect upon the symptom, and (2) the nonrecurrence of the symptom after a therapeutic trial. Continued treatment with substances of the proper group indicated that I had controlled the symptom entirely or markedly decreased the intensity of the recurrence in most cases. In some cases, the cessation of treatment after an interval of time was not associated with symptom recurrence. In other cases, recurrence took place at long intervals after the discontinuance of treatment, and the symptom responded to the same method of treatment. In the latter group, a reevaluation of the character of the symptom was usually made to determine whether the recurrent attack of vertigo had the same character, either alkaline or acid, as the original attack. If a change in character occurred, appropriate treatment with the proper group of substances afforded relief from the disturbing symptom.

Charcot's observation that the vertiginous symptom disappears spontaneously in cases of peripheral vestibular cochlear disease when the patient becomes completely deaf is common knowledge to those members of the profession who treat or see cases of vertigo. These cases of peripheral vestibular cochlear disease have a long antecedent history of difficulty in hearing and tinnitus, and it was observed that the disease (Ménière's syndrome) begins in the cochlea. In cases of vestibular disease without cochlear involvement, it would be reasonable to expect that involvement of the cochlea would ultimately ensue. In my experience, this situation has never been observed. Williams has made the observation that many cases of perceptive deafness are in reality early cases of Ménière's disease without vestibular symptoms.

Occasionally, a case is seen with a history of a recent attack of peripheral vestibular disease lasting a short time, e. g., three to four days, verified by a competent observer. Immediate examination thereafter has revealed no abnormality in hearing or in the vestibular response. The induced vestibular response is identified by the patient with the symptom that occurred at the time of his illness. This has occurred on one occasion at the Brooklyn Eye and Ear Hospital, and the patient did not respond to the substances utilized in determining the character of the symptom. The symptom of vertigo could not be induced by continued use of the alkaline or acid lipids.

The foregoing remarks were made because these factors are to be considered in determining the value of any treatment. Given the first attack of vertigo in a patient with or without cochlear involvement, Revici's method has added importance because during a first attack definite information on the

effect of the substance can be immediately ascertained. Without the patient's knowledge of the action of a substance, the symptom can be increased or decreased at will. Thus, the psychological factor can be eliminated and has no bearing on the effects of the substance. Even in a short-lived attack, the effects upon the symptom can be determined.

With the currently accepted methods of therapy, one does not get immediate results and can only conjecture whether the therapy had any influence on the symptom or whether a spontaneous remission occurred when the vertigo disappeared.

Many types are seen, ranging from the case with complete cessation of the symptom and no further recurrences having no clinical evidence that a vestibular cochlear injury had been sustained, to the case with extreme injury as indicated by the physical evidence and the repeated recurrences. The factor of the effect of a given group of substances on the symptom and a coincidental remission of the symptom cannot be entirely eliminated. In view of the immediate response of the symptom to the substances used in the majority of cases, the question whether the relief of the symptom was a coincidence rather than a response to the substance is certainly obvious.

Therefore, each case was judged on its own clinical picture. A result was considered good when the symptom was relieved after treatment and the recurrence of new attacks was infrequent or lacking during the period of observation. The result in a given case was considered unsatisfactory either when the immediate beneficial effect was present but was not entirely satisfactory for symptom relief, or when the reappearance of the attack could not be controlled or prevented.

A case that had a negative response to the administration of the two groups of substances was not included in the results of this series, even though the case was treated for some time to find out whether a response could occur with the use of the various injectable liposides that Revici has developed.

REPORT OF CASES

Case 6.—A white married woman, a housewife aged 34, was first seen by me on Dec. 19, 1947. She

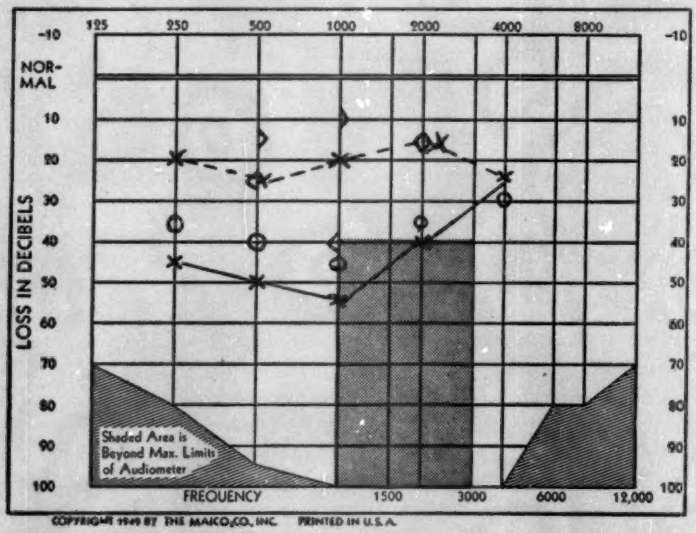

Fig. 1 (Case 6).—Solid line indicates air-conduction audiogram of left ear taken May 1, 1948, before the fenestration operation. Broken line indicates air-conduction audiogram of same ear (left) three months later, Aug. 21, 1948. Hearing improvement was maintained until vertigo and roaring tinnitus started in left ear. Preoperative bone-conduction audiogram is shown. X, left ear; O, right ear. (From Ear, Nose, and Throat Service, Greenpoint Hospital)

had bilateral deafness of 10 years' duration. Her sister was hard of hearing and was now wearing a hearing aid. A general physical examination was negative. There was no tinnitus. The patient had two children and claimed that the hearing had become worse with each delivery. She had an average hearing loss of 40 db. in the right ear and of 48 db. in the left ear for the three speech frequencies (500, 1000, 2000 cps). The bone-conduction test showed an average loss in the right ear of 13 db. and an average loss of 26 db. in the left ear for the three speech frequencies (Fig. 1, audiogram). In the 2000 cps frequency, there was a 15 db. loss by bone conduction, giving a cochlear reserve of 25 db. The C-64 fork was not heard in either ear.

On May 3, 1948, the patient was admitted to the Ear and Throat Service of the Greenpoint Hospital. She had been pregnant for about four weeks. A left fenestration operation was performed according to the Lempert technique. Except for a moderate degree of vertigo, her postoperative recovery was uneventful. She was discharged on the 12th postoperative day with no vertigo. The subsequent postoperative result was excellent. The hearing for the speech frequencies was above the 30 db. level: 500 cps = 25 db., 1000 cps = 20 db., and 2000 cps = 15 db. The 2000 cps tone attained the postoperative bone-conduction level of 15 db. During the delivery of her third baby, the hearing receded, but she recovered her postoperative level one month after delivery.

She enjoyed good hearing in the ear operated on (left) for almost four years, until Aug. 3, 1952, when she complained of a continuous roaring noise in the ear. Shortly thereafter, she complained of severe vertigo with a tendency to fall toward the left. The severe attacks occurred two or three times a week, but the dizziness persisted and would increase on sudden change of position of the head and on getting out of bed in the morning. Since the onset of this illness, she also complained of frontal headache which radiated from the left to the right side. The headache and the vertigo were worse at night.

On Sept. 4, examination showed a well-healed left endaural cavity. Application of a cotton pledget at the fenestration site accentuated the dizziness. The audiogram showed a depression for all frequencies, with an average loss in hearing for the speech frequencies of 50 db. in the right ear and of 45 db. in the left or fenestrated ear. Except for a swaying to the left on standing, no other contributory signs were present.

It was suspected that her symptom was alkaline because it usually became worse at night. This was confirmed by the administration of nikethamide solution, 1.5 cc., intramuscularly. This medication relieved her vertigo, which was present during the examination. She was placed on a schedule of 7% *n*-butanol solution, 15 drops q. i. d. and oftener, to control the symptom of vertigo. In addition, she received 1 cc. of the nonsaponifiable fraction of human placenta in oil intramuscularly each week

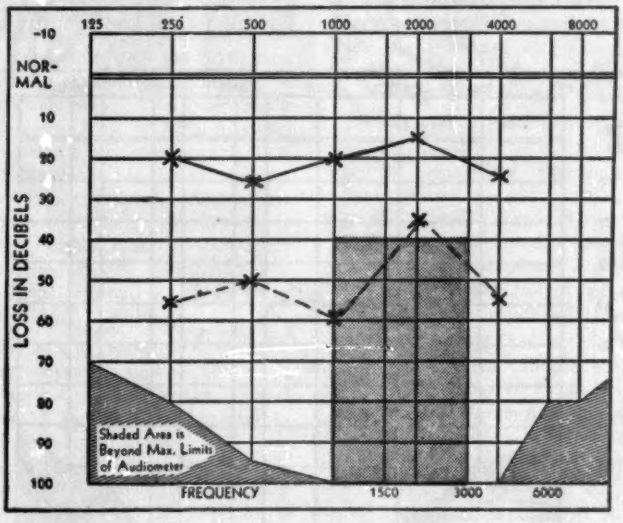

Fig. 2 (Case 6).—Solid line indicates postoperative air-conduction audiogram three months after fenestration operation. This hearing level was maintained for three and a half years, until onset of tinnitus and vertigo. Broken line indicates recession in hearing that took place five weeks after onset of vertigo, Nov. 19, 1952. X, left ear.

for three weeks. By Oct. 5, the symptom of vertigo was controlled. The tinnitus was less intense; the headache and impairment of hearing persisted. At this time, the injectable material was changed, and she received 1 cc. of the nonsaponifiable extract of lymph nodes (cattle) in oil by intramuscular injection at weekly intervals. By Dec. 5, all symptoms had disappeared; the dizziness had not returned since Oct. 5. A low inconstant tinnitus remained. On Jan. 17, 1953, she was given 1 cc. of 5% xanthophyll (extracted from spinach) in oil. This was repeated at intervals of three weeks for three injections. All medication was discontinued on Feb. 14. She was last seen on March 30, 1953. She had received or taken no medication since her last visit. There was no return of symptoms. The hearing had receded to below the 30 db. line, and she was wearing a hearing aid (Fig. 2, post-treatment audiogram).

Comment.—This case is presented because, when first seen, the patient had a hearing difficulty of 10 years' duration. This was diagnosed as due to clinical otosclerosis. The fenestration operation afforded her practical hearing for more than three and a half years. Then, there was superimposed on this condition the symptom complex of peripheral vestibular cochlear disease (endolymphatic hydrops). The latter condition was promptly relieved by the administration of *n*-butanol solution, 7%. In addition, three substances were given: the nonsaponifiable extracts of human placenta, lymph nodes from cattle, and xanthophyll in oil. These substances used interchangeably in other cases in this series had a beneficial effect on an alkaline symptom of vertigo. Here it is interesting to speculate whether the otosclerotic focus involving bone and the endolymphatic hydrops, which is a hair-cell disease, had not been present together over a long period. When the fenestration operation was performed, it is likely that the hair-cell disease was present and expressed itself as part of the picture of hearing impairment seen in the audiogram. This idea of the presence of deafness as an expression of Ménière's syndrome existing for a long period without the associated vertigo is in keeping with the conclusions of Williams.

On April 4, 1955, the patient was contacted by telephone. She had not taken any medication after one month on leaving my care. She was free of vertigo except for fleeting attacks, never as severe as when she first visited me. The hearing loss apparently remained stationary, and she could hear well on the phone without a hearing aid. She was not incapacitated by the momentary attacks when they appeared. The tinnitus was of low intensity and inconstant.

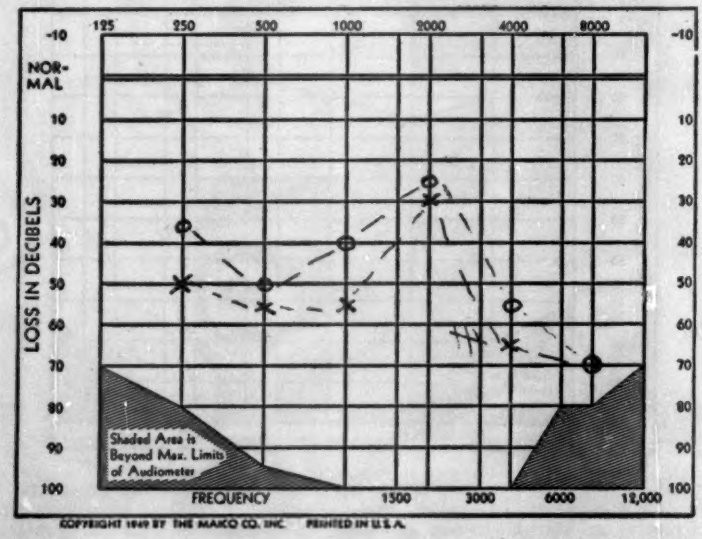

Fig. 3 (Case 6).—Audiogram two and a half years after attack of vertigo (April 16, 1955).

The headaches recurred but were mild in character. She had not sought any other treatment.

CASE 10 (Referred by Dr. Harry Burgess).—A white male chemist, aged 22, was first seen by me on Oct. 6, 1952. His chief complaint was violent dizziness on sudden turning of his head to the right, on bending, or getting out of bed quickly.

His present illness started on Aug. 5, 1952. While playing handball, he made a sudden turn to the right, became very dizzy, and fell to the ground. He thought he had blacked out. There was no hearing loss, tinnitus, headache, or vomiting. On Aug. 20, he had another short attack that was momentary. On Sept. 1, he began to have diplopia. X-ray examination of the skull and sinuses at the Brooklyn Eye and Ear Hospital, neurological consultation, and a complete physical examination disclosed no abnormality. Ophthalmological examination by Dr. Harry Burgess revealed a paralysis of the right external rectus and inferior oblique muscles to account for his diplopia. Audiograms showed no hearing loss. This confirmed the findings at another hospital at the onset of his difficulty. Nystagmus to the left of a rotating type was elicited repeatedly on his turning the head to the left or right or bending forward. Romberg's sign and past-pointing were not present. Cold caloric and galvanic tests revealed normal responses. On Oct. 8, he was given a subcutaneous injection of 1 cc. of isotonic saline solution. The symptom of vertigo was not influenced. Twenty minutes later, he was given a subcutaneous injection of 1 cc. of 4% sodium thiosulfate solution. In 20 minutes, the vertigo became unbearable and the nystagmus occurred on any change of position, i. e., turning the head, bending, or rising slowly from lying on the back. After 20 minutes, 2 cc. of 7.5% *n*-butanol solution in isotonic saline was administered intramuscularly. Relief was obtained in about 10 minutes. He was given an intramuscular injection of the nonsaponifiable fraction of bovine lymph nodes in oil. This gave him further relief in another 15 minutes. He was placed on oral administration of 7.5% *n*-butanol solution, 20 drops q. i. d. and every 15 minutes p. r. n.

By Oct. 11, the attacks occurred infrequently and were not as pronounced on turning. Five drops of glycerine in water t. i. d. was added to the oral medication. On Oct. 13, 15, and 18, the nonsaponifiable fraction of bovine lymph nodes in oil, 1 cc., was given. On the last date, he stated that he could not bring on the attacks easily. He occasionally veered to the left when walking. He continued on this schedule of oral medication. On Oct. 20, the dizziness was very much reduced. By Oct. 25, the attacks of vertigo were fleeting, and by Oct. 27, the vertigo had disappeared. The diplopia remained unchanged. From Oct. 31, he received four additional injections and the vertigo did not return. He desired to join the military service in the chemical branch and was accepted; I have not seen him since.

Comment.—This case is presented because of the unusual combination of diplopia and positional vertigo. The causative factor could not be ascertained. The case also illustrates the effect of one of the more recent complex substances that were developed by Revici and its beneficial action on an alkaline symptom.

CASE 13.—A white married woman, aged 47, was first seen by me on Jan. 9, 1953. For the previous two years she had complained of objective vertigo which came in attacks that were mild during the first six months. She then sought treatment, and for the previous one and a half years had received the usual therapy without gaining any relief from the vertigo attacks as they occurred. This treatment consisted of salt-free diets and the administration of hormones, particularly estrogen preparations.

During the past year, she had had three severe attacks, each lasting four to six weeks, and in the interim she was never free from unpredictable sensations of vertigo accompanied by nausea. The most recent attack started explosively five weeks previously, with severe objective vertigo and pressure over the left ear, as well as inconstant head noise that she thought came from the left ear. This attack was associated with nausea, vomiting, and staggering when she attempted to walk. There was no complaint of headache or hearing disturbance.

She had been carefully examined by other physicians, and no contributory findings were noted. When she came to my office, she required support because of her staggering gait. She was very apprehensive and anxious. Her hearing was normal. There was no corneal or facial anesthesia. She presented a direction-fixed rotational nystagmus to the left. During a test of her equilibrium, she fell backward on standing with her eyes closed.

She was given 1.5 cc. of nikethamide solution intramuscularly. This afforded her partial relief and was followed by an intramuscular injection of 1 cc. of a 30% solution of *n*-butanol. Within 15 to 20 minutes, she felt much better and the staggering gait was reduced to a slight swaying on walking. One-half hour afterward, she was given an injection of 1 cc. of 5% xanthophyll in oil, and a half-hour later she was able to walk about with a greater degree of comfort. Apprehension and fear had disappeared. She was given oral medication of 15 drops of 7.5% *n*-butanol solution q. i. d. and every

15 minutes p. r. n. In addition, she received 2 drops of pure glycerin in water q. i. d.

On Jan. 21, she returned; she felt slightly nauseated but had had no attacks of vertigo. She was given 1 cc. of 5% xanthophyll in oil three times a week. On Feb. 6, there was a recurrence of fullness in the left ear with headache. This was relieved by additional oral medication. On Feb. 11, she experienced a mild attack of vertigo, which was again relieved by oral medication. Administration of xanthophyll was reduced to about one injection every 10 days. On April 6, she reported freedom from attacks for seven weeks. On June 13, she had a fleeting attack which was immediately relieved by oral medication. On June 25, she went to Washington, D. C., on a business trip. She was very active but did not experience vertigo. There were no attacks after this, and medication was discontinued. On Dec. 5, she came in during an attack, received an intramuscular injection of 10% solution of the nonsaponifiable fraction of butter in oil, and was relieved in about 20 minutes. This medication was given at weekly intervals until Jan. 1, 1954. She now returns every three to four months for observation. Her attacks recur at intervals of months, are very mild, and are relieved by oral medication.

Comment.—This case illustrates the result obtained on persistent treatment of vestibular disease by the classical methods over a long period of time. This patient was an active woman and was understandably anxious and annoyed when her activities were curtailed. These reactions promptly vanished when she realized that the attacks could be controlled and, furthermore, could be attenuated in intensity and duration when they did occur. This patient is in marked contrast to a patient with all the signs and symptoms of peripheral vestibular disease who, in addition to the feeling of anxiety, presents a marked psychogenic overlay based on personality changes that have existed for a long time prior to the onset of vertigo.

Such a patient is very difficult to treat because the psychosomatic overlay acts in some cases in the same way as an active distant focus of infection, i. e., gall-bladder disease or an abscessed tooth, the toxic products of the abnormal metabolism continuously activating the abnormal labyrinthine focus through the blood stream. I have reported cases such as these in my earlier article on vertigo.

In addition to the foregoing, this case is of interest because the nonsaponifiable fractions from three different sources were utilized: (*a*) from butter; (*b*) from carp (fish intestine), and (*c*) xanthophyll extracted from spinach. Revici developed these substances in an endeavor to produce a substance with greater intensity of effect. They all belong to the broad group he has designated as lipobases. From my own observations, all these substances have the same effect as 7.5% *n*-butanol solution, but their action is more intense and more sustained.

As of the date of writing, April 16, 1955, the patient has not had any attacks for about seven months, is still under observation, and has had no additional medication. She occasionally experiences fulness in the left ear which signifies the onset of a vertigo attack, and she takes oral medication to ward off the attack.

Case 17.—A white man, aged 54, was first seen by me on Sept. 13, 1952. His occupation was laundry truck driver. He was carried into my office during an acute severe attack of vertigo associated with nausea and vomiting. He could not stand without support. The attack had started about 24 hours prior to my seeing him. This was his third severe attack in four weeks. The first attack had been ushered in with a similar series of events.

He had mild diabetes, well controlled by diet, and was taking no insulin. The diabetes was first brought to his attention five years before. He also had a gastric ulcer.

His present illness started with inconstant tinnitus in his right ear four years previously. After an interval, it became constant and he noticed an impairment of hearing in that ear. At that time, he was aware of fleeting attacks of vertigo that would pass off without treatment. Four weeks before, he had his first severe attack lasting several days. He had received dimenhydrinate (Dramamine), histamine, and nicotinic acid without relief. He had also been given sulfisoxazole (Gantrisin).

Examination showed nystagmus to the right. The audiogram revealed a perceptive loss in the right ear by both bone and air conduction. For the speech frequencies, the right ear presented a 65 db. loss by air conduction and a total loss by bone conduction. The left ear showed an average loss for the speech frequencies of 9 db. by air conduction and of 20 db. by bone conduction.

A physical examination, including an evaluation of his diabetic condition, revealed nothing of sig-

nificance. The blood pressure was 130/80. The galvanic and caloric tests were normal.

An injection of nikethamide solution, 1.5 cc., immediately intensified the vertigo and the nausea. Twenty minutes later, an injection of 2 cc. of 4% sterile sodium thiosulfate solution subcutaneously immediately lessened the intensity of the vertigo. From these observations, it was considered an acid symptom, according to Revici's classification. The patient was placed on a schedule of 15 drops of 10% solution of sodium thiosulfate q. i. d. and oftener if necessary for the relief of attacks of vertigo. In addition, he received 1 drop of sulfonated oil three times a day. He was given an intramuscular injection of 1 cc. of the conjugated fatty acids from cod liver oil with sulfur once or twice a week. A diet for patients with acid symptoms was also prescribed.

On this regimen, he was able to continue his occupation as a driver of a laundry truck. The attacks continued to occur on and off but with decreasing intensity. He carried the bottle of the 10% solution of sodium thiosulfate constantly while at work and took several doses when he felt an attack of vertigo coming on. By Oct. 25, he complained only of fleeting attacks of vertigo that were promptly relieved with several doses of 20 to 40 drops of 10% solution of sodium thiosulfate. The injections of the conjugated fatty acids from cod liver oil with sulfur were continued at weekly intervals. By Jan. 3, 1953, he had had complete freedom from attacks of vertigo for three weeks. He had a fleeting attack that morning which was relieved by the medication. The injections had now been given every two weeks. As the symptoms subsided, the oral medication was continued and he received injections of the conjugated fatty acids with sulfur at monthly intervals. From March 14 to April 11, he had only two mild attacks of vertigo that were relieved by the oral medication. From April 11 to Sept. 5, he received eight injections of the conjugated fatty acids with sulfur at fortnightly or monthly intervals. During this time, he had no oral medication. On Sept. 4, he experienced a moderate attack that was relieved by 10% sodium thiosulfate solution, 15 drops every 15 minutes for four doses. From Sept. 11 to 25, he had fleeting attacks of vertigo which were not relieved by the medication, and on Sept. 25 he had a very severe attack associated with vomiting, headache, and increased tinnitus. The medication gave no relief, and it was the patient's opinion that the vertigo became worse. This usually occurs when the character of the symptom changes from acid to alkaline, and vice versa. On Sept. 26, he was given 1 cc. of a sterile solution of 7% *n*-butanol by intramuscular injection. This relieved the headache in about 15 minutes. An injection of 1 cc. of dihydroergotamine had a similar beneficial effect. It did not provoke the symptom of vertigo. He was then given 1.5 cc. of nikethamide by injection. After a half-hour, there was no sign of vertigo. Subjectively, he felt much better. Again after a half-hour, he received 1 cc. of the nonsaponifiable fraction of butter intramuscularly. After further observation for a half-hour, the patient stated that there had been progressive improvement with each injection. Vertigo had not been provoked. He was placed on a schedule of oral administration of 7% *n*-butanol solution in water, 15 drops q. i. d. diluted in a little water. On Sept. 28, an audiogram showed no change in his hearing by air conduction. The hearing in his right ear had not become worse. The tinnitus in the right ear and the headaches had been severe during the previous two days. He also had had mild attacks of vertigo, but these were relieved by *n*-butanol taken orally every 15 minutes. He was given an intramuscular injection of 1 cc. of 30% *n*-butanol in water, with sodium benzoate added to increase the solubility of *n*-butanol in water. The oral medication was increased to 15 drops of *n*-butanol in water every three hours. His diet was changed to one for alkaline symptoms. On Oct. 3, he stated that he felt much better and had only slight fleeting attacks of vertigo with the medication.

He was given an injection of 1 cc. of the nonsaponifiable fraction of butter every two weeks. The diabetic status was checked by Dr. Harry Levine. At this time, his blood sugar had risen to 200 mg. per 100 cc. There was no acetonuria. The urinary sugar was 2.5%. When seen on Nov. 13, he had had an abscessed tooth removed. The diabetes was controlled. There had been no attacks of vertigo. His headaches recurred from time to time and were controlled by acetylsalicylic acid, 5 grains (0.3 gm.). On Dec. 26, he had the first mild attack of vertigo in two weeks, which was relieved by oral medication. He remained well until May 22, 1954, when the vertigo returned and the medication gave him questionable relief. The medication made his condition worse, and the vertigo changed from an increase in intensity in the evening to an increase in intensity in the morning. At this time, my notes show that Hydergine had a beneficial effect on the vertigo, along with sodium thiosulfate solution and sulfonated oil. Dihydroergotamine had an adverse effect. The metabolic imbalance had again changed and with it the character of the symptom. On May 29, he was given sodium thiosulfate solution, 10 drops q.i.d., and sulfonated oil, 1 drop t.i.d., orally and an injection of 0.5 cc. of the conjugated fatty acids with sulfur in oil. On June 12, a similar injection was given. I did not see him again until Aug. 7, when he had a severe attack. By Aug. 9, this attack was controlled with oral medication in increased dosage. During this attack when the

vertigo was active, I gave him 0.5 cc. of the saponifiable fraction of the intestine of pig in oil intramuscularly. This controlled the attack of vertigo in 20 minutes. It was again given on Aug. 18, Sept. 12, and Oct. 16. Oral medication was discontinued, and he has remained free of attacks to date, April 10, 1955. He comes in every six weeks for an injection of a lipoacid. An attack of virus infection with elevation in temperature lasting three weeks did not bring on the symptom of vertigo.

Comment.—This case has been abstracted at some length because it serves to illustrate what can be accomplished with this method in a protracted case. In addition, the patient was a diabetic and presented a high blood sugar during one of his attacks. This could adversely influence the outcome. Then, he developed an abscessed tooth. Prior to its extraction, the diseased tooth, through its abnormal metabolism, could adversely influence the primary lesion in the inner ear and bring on the vertigo attacks. The lesion changed its character from acid to alkaline, and then back again to acid. The attack of virus infection with elevation in temperature did not provoke any attack of vertigo because, being an alkaline lesion liberating fatty acids, it served to neutralize the inner ear lesion which, at the time of the virus infection, was in an acid stage and was liberating sterols. The activity of the generalized infection served to neutralize the effects of the local inner ear lesion, probably through the acid-base balance. When the character of the vertigo changed, the medication could be altered to meet the changed metabolic process and so control the attack.

This case is of interest also from the point of view of the substances utilized. The more complex substances extracted from the tissues of pig and carp intestine, conjugated fatty acids with sulfur, acted in the same manner as the simple substances, such as *n*-butanol, sodium thiosulfate, or sulfonated oil. The intensity of the effect was greater with the complex substances. This phase of the investigation was brought out in my article on head and neck pain. I have given a summary of the newer substances that were developed by Revici earlier in this article.

A further point of interest that I wish to stress is the fact that this patient has never been hospitalized from Sept. 13, 1952, the day I first saw him, to the present, April 10, 1955, approximately two and a half years. All procedures were done in the office. It was never necessary to treat the patient at home. He remained at his work, which demanded a high degree of activity, such as driving a laundry truck, delivering laundry, getting in and out of his truck, and going up and down stairs. The intense anxiety he felt at the beginning of his illness because he believed it would be impossible to support his wife and children rapidly ameliorated when he became confident that the attacks, when they did occur, would be controlled by simple oral medication. This feeling of anxiety changed to one of only mild annoyance when the attacks did take place. During this long period, I had been tempted to perform a labyrinthotomy, in the hope of relieving his attacks of vertigo, but, happily, I did not do so and am gratified with the result as it stands.

Case 20.—A white longshoreman, aged 44, was first seen by me on May 13, 1953. He complained of repeated attacks of vertigo and staggering while at work on the docks and in ships for the past two years. For about a year prior to the onset of vertigo, he noted an inconstant tinnitus in both ears and a progressive deterioration in hearing. The latest attack had been on May 8, 1953.

Examination revealed a powerful, well-built man with no contributory findings pertinent to his complaint. He was very anxious about his condition because his ability to earn a living in his occupation was impaired. During the previous two years, he had received several courses of therapy with nicotinic acid and histamine, which afforded no relief. The noise at the docks and on the ships made him irritable, and during an attack he was forced to lie down and try to take refuge in sleep. Lately, he had been taking sedatives, but they were of no help.

The audiogram revealed an average 78 db. loss of hearing in his right ear and a 58 db. loss in his left ear for the speech frequencies. There was no bone conduction in either ear. The galvanic and cold caloric tests showed hypoirritability of both labyrinths. There was no past-pointing; the Rom-

berg sign was not elicited. No vertigo was present. The Eustachian tubes were patent.

Tests with acid and alkaline electrolytes by mouth and *n*-butanol and sodium thiosulfate by mouth and injection produced no result. I could not elicit the symptom of vertigo. For one month, he took sulfonated oil, 3-4 drops t. i. d., sodium thiosulfate solution, 15 drops q. i. d. In addition, he was given a weekly injection of 1 cc. of the conjugated fatty acids of cod liver oil with sulfur for four doses. This medication had no effect on the tinnitus or the hearing difficulty. It did not elicit the symptom of vertigo.

On May 6, he was placed on oral medication consisting of 20 drops of 7.5% *n*-butanol q. i. d. and 5 drops of glycerine t. i. d. and was given an intramuscular injection of the nonsaponifiable fraction of butter at weekly intervals for four weeks. At the end of one month, he felt no better or worse. On July 10, he had an attack of vertigo. He received 1 cc. of nikethamide solution for two doses at 20-minute intervals and fell asleep. I have not seen him since.

Comment.—This case illustrates authentic peripheral vestibular cochlear disease in which acoustic trauma could well be a factor. The patient was a longshoreman, and excessive noise on the docks and in the holds of ships was one of the hazards of his work. His disease could well have been superimposed on or concomitant with labyrinthine hydrops. The case illustrates Charcot's observation of many years ago that the vertigo associated with peripheral vestibular cochlear disease is ultimately arrested with the onset of extreme deafness. I cannot believe that the two doses of nikethamide he took for the relief of his attack on July 10, 1953, were anything but coincidental. During the two months he was under observation, I did not observe nor could I elicit the symptom of vertigo. This further confirms Revici's hypothesis that an active pathological focus must be present, the abnormal metabolism of which must deviate to either the alkaline or the acid side, in order for the substances utilized to be active.

CASE 34.—A white married woman, aged 38, a housewife with two children, was first seen by me on Nov. 7, 1953. She complained of vertigo of four weeks' duration, during which time she was confined to bed because of its severity. This was her third attack, the first one occurring in August, 1952, associated with left acute otitis media, and lasting 10 days, for which dimenhydrinate and penicillin gave relief. She continued ambulatory. Six months later, she had another attack lasting a few days. This third attack was so severe that she had to go to bed.

She first noticed that when driving her car, about five weeks previously, she was impelled to turn the car to the right and compensated by having to pull it to the left. This was accompanied by lightheadedness. Severe vertigo of a subjective type followed. Buzzing in her ear (left) accompanied the vertigo and was inconstant. When lying in bed on her left side, she felt as if some force were pulling her out of bed. Headache occurred in the left temple. When reaching for a glass of water, she was uncertain. Hearing in the left ear had been poor for many years following an attack of otitis media as a child. Left simple mastoidectomy had been performed twice from 1938.

She had received shock therapy for a depressed state; the last treatment was given in 1949. This state has not troubled her since. For the present vertiginous attack, she was advised to undergo radical mastoidectomy.

Examination showed a slightly positive Romberg sign; there was no past-pointing, nystagmus, or corneal or facial anesthesia. She was unsteady on reaching for an object and complained of vertigo. The left ear showed a dry chronic adhesive process with part of the malleus and all the stapes clearly visible. The ear drum membrane was absent. The galvanic response was normal and so was the cold caloric response in the right ear. The induced vertigo was identified as the symptom of which she complained. A cold air douche in the left ear similarly induced a sensation of vertigo and normal nystagmus. The audiogram revealed an average hearing loss for the speech frequencies in the left ear of 43 db. by air conduction and of 40 db. by bone conduction. The right ear showed an air-conduction loss of 7 db. and a bone-conduction loss of 23 db. for the speech frequencies. She was given 1 cc. of dihydroergotamine, which intensified the symptom. Shortly thereafter, she was given 1.5 cc. of nikethamide solution and then 1.5 cc. of 7.5% *n*-butanol in saline solution, intramuscularly. These did not ameliorate her symptom. She was then given 1 cc. of 4% sterile solution of sodium thiosulfate subcutaneously. Within 20 minutes, this had a beneficial effect in the amelioration of her symptom. She was given oral medication of 15 drops of 10% sodium thiosulfate solution q. i. d. and 3 drops of sulfur in oil t. i. d. In addition, she received an intramuscular injection of 1 cc. of the conjugated fatty acids from cod liver oil with sulfur three times a week. By Nov. 7, she felt well enough to drive her car. On Nov. 16, she was working in the garden. The exercise induced a fleeting attack of vertigo lasting seconds. In spite of an intercurrent

cold on Nov. 30, lasting three weeks, she continued up and about with only very transient attacks of vertigo. On Dec. 12, it was apparent that the acid symptom was changing its character and was becoming alkaline. Revici has pointed out that this can be one of the characteristics of the evolution of a pathological focus.

On Dec. 15, the medication did not help her and she discontinued it herself, feeling much better on Dec. 16. On that day, she received an injection of procaine hydrochloride for dental work. This did not bring on the dizziness. The medication was now changed to 7.5% *n*-butanol solution, 10-12 drops q. i. d. orally, and 1 cc. of the nonsaponifiable fraction of butter at weekly intervals. This schedule of medication immediately controlled the vertigo, and on Jan. 6, 1954, she was able to drive the car, taking turns with her husband on a trip to Florida. She did not feel any sensation of vertigo on the trip. She continued to take *n*-butanol orally for several days, then all medication was discontinued. From Jan. 27 to July 9, she had no symptoms. She then had fleeting attacks at infrequent intervals, which were relieved by oral administration of *n*-butanol solution. A bad attack lasting two to three hours occurred on Aug. 30. At that time, she was given 1 cc. of the nonsaponifiable fraction of carp intestine in oil intramuscularly. This was another substance developed by Revici which was being utilized at this time to control alkaline symptoms. This substance effectively controlled the attacks. It was given every three weeks. On Oct. 1, she telephoned to say that the vertigo had recurred the previous night with great intensity. She was very apprehensive about my being able to control the attack. With the galvanic current test and the cold caloric test in the right ear, I was able to reproduce the symptom of vertigo and the associated nystagmus. She immediately stated that this was not the symptom she had experienced. On subsequent questioning, she admitted that she had had an emotional upset and developed a feeling of confusion and tenseness she thought was the onset of an attack of vertigo. She had had no vertiginous attacks to April 12, 1955, when I contacted her by phone. On that day, she had fleeting attacks which were controlled by taking orally 25 drops of *n*-butanol q. i. d. An audiogram on Nov. 20, 1954, showed in the left ear an average air-conduction loss of 42 db. and a bone-conduction loss of 10 db. for the speech frequencies. The right ear had an average air-conduction loss of 10 db. and an average bone-conduction loss of 6 db. for the speech frequencies.

Comment.—This case is presented because it illustrates four points of interest. The patient was under continual observation from Nov. 7, 1953, to April 13, 1955. She presented a hearing defect in the left ear due to a chronic adhesive process that had existed from childhood. She developed a depressive state for which shock therapy was given 17 years previously. An intercurrent episode of emotional tension developed during the course of treatment that was identified with real vestibular vertigo. The treatment is of interest because several different complex substances developed by Revici of the acid and nonsaponifiable fractions of lipids from various sources were effective in controlling her symptom when it recurred. In spite of her bilateral hearing defect, which was greater on the left with associated bone-conduction loss and tinnitus indicating a perceptive loss in the left ear, the hearing in this ear returned to normal by bone conduction, while the air-conduction loss was greater for the low tones one year after treatment was started, indicating a conductive type of loss. This loss is consistent with a chronic adhesive process. The right side showed no hearing loss one year after treatment was instituted. After each attack, it was expected that the hearing would not improve, especially after the third and severest attack. In this case, a favorable outcome was obtained for the patient's hearing. The tinnitus also disappeared.

The question of operating on the left ear was seriously considered. A radical mastoidectomy with probable labyrinthotomy had been proposed. For the past eight years, I have advised that such a procedure might be necessary if lipid therapy should fail. But I have never had occasion to carry out the operation.

Case 37 (Referred by Dr. John Galbraith).—A white woman, aged 67, was first seen by me on Nov. 20, 1953. She complained of attacks of vertigo of four to five years' duration. The attacks were preceded by tinnitus in the right ear and bilateral impairment of hearing. Following these symptoms in 1948, there occurred a sudden attack of objective vertigo associated with nausea and vomiting. Sudden movement would bring on subsequent attacks. There was no headache but a constant awareness of "something wrong" in her right ear. She was given a salt-free diet, ammonium chloride orally, and nicotinic acid injections. She thought she had

some relief after the first injection of nicotinic acid. This treatment was carried out for two years but gave her relief for only six months. Emotional upsets were associated with the onset of the attacks. She did not believe that the treatment over a period of two years with the above substances curtailed or ameliorated the attacks when they occurred. A fenestration operation through the postauricular route, with destruction of the right labyrinth, was performed in March, 1952, at another hospital. This procedure afforded relief from attacks of vertigo for one year. Since then, the attacks of vertigo had recurred "once a week or once a month." Apparently no drug had afforded her relief. Her physician believed that emotional upsets brought on the attacks.

Audiometric examination showed a total loss of hearing in her right ear. All sounds presented to her right ear were referred to her left ear, which showed an average loss of 65+ db. for the speech frequencies. Tests with the 512 and 1048 forks showed all sounds referred to the left ear. The C-4 fork was not heard in the right ear but was heard in the left ear. She swayed markedly on standing with her eyes closed. The galvanic test with 4 ma. of current to both mastoids induced an objective vertigo which she identified as identical with the spontaneous attacks of vertigo. There was no spontaneous nystagmus or corneal anesthesia. The cold caloric test induced dizziness but no nystagmus on the right side. The same test on the left ear showed hypoirritability.

She did not respond to the electrolytes administered orally, either acid or alkaline. Nor was any response obtained with the lipids given orally or by injection. She was last seen by me on Nov. 28, 1953.

Inquiry to Dr. Galbraith revealed that the attacks of vertigo are becoming less frequent, and the patient is receiving no particular medication for the relief of the symptom. He believes that emotional tension states were a large factor in bringing on these attacks.

Comment. — This case again illustrates Charcot's observation that Ménière's disease tends to improve when the progression of the labyrinthine disease has culminated in total loss of hearing. The psychosomatic overlay was probably a factor in this case.

CASE 41.—A white male bartender, aged 38, was first seen by me on Feb. 27, 1954. His principal complaint was sudden violent attacks of vertigo that had occurred with increasing frequency for the past 10 years. He stated that his present complaints started with tinnitus in his right ear about 10 years ago. This was constant and roaring in character and persisted for about one year, with gradual impairment of hearing on the right side. In the past two years, he noticed an occasional sense of fulness in his left ear, with associated difficulty in hearing. Then, the tinnitus suddenly increased in intensity and was followed by a violent attack of vertigo, objective in type, accompanied by nausea, vomiting, sweating, and a right-sided headache. This attack lasted several days but was followed by recurring attacks which gradually became more intense and more frequent in the past year. He could not continue to work as a bartender because of the constant moving about associated with his duties. Finally, he went to the hospital, where the diagnosis of Ménière's disease was made. He received a thorough survey during his stay of eight weeks in the hospital. There were careful physical, neurological, and allergy examinations, including skin tests, and x-rays of the skull, a gastrointestinal series, and an electroencephalogram were made. A psychiatric survey indicated that he had hypochondria with gastrointestinal symptoms. Blood studies and the examination of the spinal fluid were negative. He was given histamine acid phosphate intravenously. This made him worse and brought on a severe attack. After this attack subsided somewhat, he was given nicotinic acid injections. These flushed his face and skin and afforded only very transient relief. He was sent home with instructions to take a salt-free diet with ammonium chloride. There was no relief, and the attacks of dizziness returned just as frequently.

My examination showed that he presented an essentially normal air-conduction curve in his right ear, with an average 22 db. hearing loss for the three speech frequencies. His left ear showed an average loss of 40 db. by air conduction for the speech frequencies. The C-64 fork was heard in both ears by air conduction. The Schwabasch reaction was normal. On the Weber test the sound was referred to the left ear, and the pure tones on the audiometer were referred to the left side. Recruitment was present in all the tones for the speech frequencies in the right ear.

There was no nystagmus, past-pointing, or corneal anesthesia. Both caloric and galvanic tests of the labyrinth were within normal values. The diagnosis was left peripheral vestibular cochlear disease.

He was given a subcutaneous injection of 2 cc. of 4% sterile sodium thiosulfate solution. In about 25 minutes, he became dizzy and the tinnitus in his right ear became more intense. He was then given 1.5 cc. of nikethamide solution by intramuscular injection. This relieved his dizziness shortly, and the tinnitus became less intense. He was placed on 15 drops of 7.5% *n*-butanol solution q. i. d. and an appropriate diet for an alkaline lesion. In addition, he was given, by intramuscular injection, 1 cc. of the nonsaponifiable extract of the intestine of pig twice weekly. By March 8, the dizziness had not

TABLE 1.—*Summary*

Case No.	Date	Sex	Age, Yr.	Race	Occupation	Duration	Vertigo	Result	Tinnitus	Result	Hearing Loss
1	2/14/51	F	27	N	Nurse	6 mo.	+	G	A. S.	N	A. S.
2	2/26/51	M	30	W	Laborer	2 wk	+	G	A. D.	N	Both
3	3/ 7/51	M	62	W	Laborer	6 yr.	+	G	0	0	0
4	3/ 8/52	F	35	W	Housewife	2 mo.	+	G	A. S.	G	Both
5	7/11/52	M	60	W	Laborer	2 mo.	+	G	A. S.	Imp.	Both
6	9/ 3/52	F	39	W	Housewife	5 wk.	+	G	Both	G	Both
7	9/22/52	F	68	W	Housewife	30 yr.	+	G	A. D.	Imp.	Both
8	10/ 6/52	F	66	W	Housewife	2 mo.	+	G	A. S.	G	A. S.
9	10/ 5/52	F	55	W	Housewife	7 mo.	+	Uns.	A. D.	N	Both
10	10/ 6/52	M	22	W	Chemist	2 mo.	+	G	0	0	0
11	12/ 6/52	M	43	W	Executive	15 yr.	+	G	0	0	0
12	1/10/53	M	52	W	Clerk	2 wk.	+	G	0	0	Both
13	1/19/53	F	47	W	Publicist	2 yr.	+	G	0	0	0
14	2/ 9/53	F	49	W	Housewife	2 yr.	+	G	Both	0	Both
15	2/18/53	F	59	W	Housewife	2 yr.	+	G	A. D.	G	Both
16	3/18/53	M	40	W	Plumber	1 yr.	+	G	0	0	0
17	3/18/53	M	54	W	Truckman	5 yr.	+	G	A. D.	N	A. D.
18	3/25/53	F	62	W	Housewife	3 mo.	+	G	A. S.	G	A. S.
19	4/18/53	M	62	W	Laborer	1 day	+	G	A. S.	I	A. S.
20	5/15/53	M	44	W	Laborer	2 yr.	+	N	Both	N	Both
21	4/24/53	M	51	W	Truckman	3 yr.	+	G	Both	N	0
22	5/25/53	F	64	W	Housewife	4 mo.	+	G	A. D.	G	A. D.
23	5/25/53	M	67	W	Painter	2 yr.	+	G	A. S.	I	Both
24	5/27/53	F	52	W	Housewife	1 mo.	+	G	A. D.	I	Both
25	5/29/53	M	48	W	Laborer	1 yr.	+	G	0	0	Both
26	6/ 4/53	M	42	W	Lawyer	1 wk.	+	G	0	0	0
27	6/17/53	F	43	W	Housewife	8 mo.	+	G	A. D.	N	A. D.
28	6/24/53	F	55	W	Dresses	6 wk.	+	Uns.	Both	0	0
29	7/15/53	M	50	W	Laborer	6 yr.	+	Uns.	A. S.	N	A. S.
30	8/18/53	M	58	W	Furs	4 yr.	+	G	A. S.	I	Both
31	10/ 2/53	F	35	W	Housewife	3 wk.	+	G	0	0	Both
32	10/30/53	F	53	W	Housewife	6 mo.	+	G	Both	G	0
33	11/ 1/53	F	62	W	Operator	8 mo.	+	G	Both	I	Both
34	11/ 7/53	F	38	W	Housewife	4 wk.	+	G	A. S.	G	A. S.
35	11/11/53	F	54	W	Housewife	3 yr.	+	G	0	0	0
36	11/14/53	M	22	W	Accountant	3 wk.	+	G	0	0	0
37	11/20/53	F	67	W	Housewife	5 yr.	+	N	A. D.	N	Both
38	11/25/53	F	47	W	Housewife	3 yr.	+	G	0	0	0
39	12/11/53	M	32	W	Businessman	2 yr.	+	G	0	0	0
40	1/ 6/54	M	44	W	Salesman	15 yr.	+	Uns.	A. D.	Uns.	0
41	2/27/54	M	38	W	Buyer	10 yr.	+	G	A. D.	G	A. D.
42	3/12/54	F	63	W	Housewife	2 wk.	+	G	0	0	0
43	5/ 7/54	F	56	W	Housewife	6 yr.	+	G	A. D.	G	Both
44	5/ 4/54	F	48	W	Housewife	8 yr.	+	G	A. S.	Uns.	A. S.
45	5/ 5/54	M	73	W	Laborer	8 mo.	+	Uns.	A. S.	Uns.	Both
46	6/12/54	F	62	W	Housewife	10 yr.	+	G	Both	G	Both
47	7/21/54	F	58	W	Housewife	4 yr.	+	G	A. S.	Uns.	A. S.
48	1/ 5/54	F	53	W	Housewife	2 mo.	+	G	A. S.	Uns.	Both
49	4/ 5/54	F	41	W	Housewife	1 mo.	+	G	0	0	0
50	6/20/53	M	43	W	Executive	8 mo.	+	G	A. D.	G	A. D.
51	12/24/52	F	45	W	Housewife	10 days	+	G	0	0	0

* No response to lipoacid or lipobase.

P. V. D., peripheral vestibular disease; P. V. C. D., peripheral vestibular cochlear disease; L. A., lipoacid; L. B., lipobase; G, good response; Imp., improved; Uns., unsatisfactory response; N, no response to either lipoacid or lipobase.

recurred. On March 9, the tinnitus was of low intensity, and he was much improved emotionally. He promptly returned to work. On May 5, he had a slight attack of vertigo associated with diarrhea. He ascribed this to exposure to draft. This attack promptly subsided with oral medication. The injections were now given every two weeks. On May 14, he complained of a "funny feeling" but experienced no dizziness. By June 12, the tinnitus had disappeared. On June 28, he suffered a severe sunburn, with return of tinnitus but no dizziness. He worked in a small semicircular bar at a restaurant, where I observed him turning continuously several times a minute, almost a complete 180-degree turn, but with no sign of discomfort. He now came at monthly intervals for observation and an injection of the nonsaponifiable extract of pig intestine. In Nov., 1954, he received a severe emotional shock caused by the untimely death of his oldest son. As a rule, emotional upsets brought

of Cases of Vertigo

Result	Headache	Result	Diagnosis	Treatment	Observation Time	Autonomic Symptoms	Psychosomatic Overlay	Other Treatments	Change of Lesion
N	A. S.	G	P. V. D.	L. A.	6 wk.	Nausea, vomiting	Fear	+	0
N	0	0	P. V. C. D.	L. A.	4 days	0	0	0	0
0	Vertex	Uns.	P. V. D.	L. B.	10 days	0	0	+	0
N	A. S.	G	P. V. C. D.	L. B.	24 days	0	0	0	0
N	A. S.	G	P. V. C. D.	L. B.	7 days	Nausea, vomiting	Fear	+	0
N	A. S.	G	P. V. C. D.	L. B.	6 mo.	0	Fear	+	0
N	Vertex	G	P. V. C. D.	L. A.	2 yr.	0	0	+	0
N	A. S.	G	P. V. C. D.	L. B.	4 mo.	Perspiration	0	+	0
N	A. D.	N	P. V. C. D.	L. B.	5 wk.	Nausea	Anxiety	+	0
0	0	0	P. V. D.	L. B.	6 wk.	0	0	+	0
0	0	0	P. V. D.	L. B.	5 mo.	0	0	+	0
N	Frontal	G	P. V. C. D.	L. B.	5 wk.	0	0	+	0
0	0	0	P. V. C. D.	L. B.	2 yr.	Nausea	Anxiety	+	0
0	Migraine	N	P. V. C. D.	L. A.	2 yr.	Nausea	0	+	0
0	A. S.	G	P. V. C. D.	L. A.	5 mo.	0	0	+	0
0	0	0	P. V. D.	L. B.	3 mo.	Nausea	0	+	0
N	A. D.	G	P. V. C. D.	L. A. & L. B.	2¼ yr.	Nausea	Anxiety	+	+
N	0	0	P. V. C. D.	L. A.	10 mo.	Nausea	Anxiety	0	0
N	A. S.	G	P. V. C. D.	L. B.	18 mo.	Nausea	Anxiety	+	0
N	0	0	P. V. C. D.	L. B. & L. A.*	2 mo.	Nausea	Anxiety	+	0
0	Both ears	G	P. V. C. D.	L. B.	2 mo.	Nausea	Tension	+	0
N	0	0	P. V. C. D.	L. B.	2 wk.	0	0	+	0
N	A. S.	G	P. V. C. D.	L. B.	3 wk.	0	0	+	0
N	0	0	P. V. C. D.	L. B.	6 mo.	Nausea	Anxiety	+	0
N	0	0	P. V. C. D.	L. B.	6 wk.	0	0	+	0
0	0	0	P. V. D.	L. B.	2 wk.	0	Anxiety	+	0
N	0	0	P. V. C. D.	L. A.	.....	Nausea	Anxiety	+	0
0	Frontal	N	P. V. C. D.	L. A. & L. B.	8½ mo.	0	0	+	+
N	Frontal	N	P. V. C. D.	L. A. & L. B.	2 mo.	0	0	+	+
N	Frontal	G	P. V. C. D.	L. A. & L. B.	4 mo.	Nausea, vomiting	Anxiety	+	+
G	+	G	P. V. C. D.	L. B.	5 wk.	Nausea	Anxiety	+	0
0	+	G	P. V. D.	L. B.	3 wk.	Nausea, vomiting	0	+	0
Uns.	+	G	P. V. D.	L. A.	6 wk.	Nausea, vomiting	Anxiety	+	0
Uns.	+	G	P. V. C. D.	L. A. & L. B.	1 yr.	Nausea, vomiting	Anxiety	+	+
0	0	0	P. V. D.	L. B. & L. A.	3 mo.	Nausea, vomiting	Anxiety	+	+
0	+	G	P. V. D.	L. B.	3 mo.	0	0	+	0
N	0	0	P. V. C. D.	L. A. & L. B.*	8 days	Nausea, vomiting	Anxiety	+	0
0	Tension	Uns.	P. V. D.	L. A.	9 wk.	Palpitation	Anxiety	+	0
0	0	0	P. V. D.	L. B.	4 mo.	0	Anxiety	+	0
0	0	0	P. V. D.	L. B.	1 mo.	Nausea, vomiting	Anxiety	+	0
Uns.	+	G	P. V. C. D.	L. B.	5 mo.	Nausea, vomiting	Anxiety	+	0
0	0	0	P. V. D.	L. B.	2 wk.	0	0	0	0
Uns.	0	0	P. V. C. D.	L. A.	4 mo.	Nausea, vomiting	Anxiety	+	0
Uns.	+	G	P. V. C. D.	L. B.	1½ yr.	Nausea, vomiting	Anxiety	+	+
Uns.	+	Uns.	P. V. C. D.	L. B. & L. A.	2 mo.	0	0	+	0
Uns.	..	..	P. V. C. D.	L. A.	3 mo.	Nausea	Anxiety	+	0
Uns.	0	0	P. V. C. D.	L. A.	4 mo.	Nausea	Anxiety	+	0
Uns.	+	G	P. V. C. D.	L. B. & L. A.	3 mo.	0	0	+	+
0	0	0	P. V. D.	L. B.	7 mo.	0	0	+	0
Uns.	0	0	P. V. C. D.	L. A.	1 wk.	0	0	+	0
0	+	G	P. V. D.	L. A.	2 wk.	Nausea	Anxiety	0	0

on vertigo. In this instance, there was no return of the symptom of vertigo. There was no change in his hearing defect on the right side.

Comment.—This case of 10 years' duration is interesting because it illustrates the impact that fundamental personality changes with their associated emotional upsets can have on an organic lesion, such as endolymphatic hydrops or, as here designated, peripheral vestibular cochlear disease. It also illustrates the rapidity with which the symptom of vertigo can be brought under control with the application of Revici's method, in this case about 10 days. Treatment was continued until Feb. 19, 1955, in the hope that some degree of permanent control could be obtained. Only further study can give the answer in this case. The patient was last seen on April 1, 1955, and has had no attacks of vertigo to date July 18, 1955.

ANALYSIS OF DATA FROM CASES

Revici's hypothesis was applied to 58 patients. They were taken at complete random and subjected to this investigation in the same manner as was described in my previous article on vertigo. Patients with the complaint of dizziness were accepted. Although when originally seen or during my observations no case presented symptoms pointing to a diagnosis of brain tumor, one such case (Case 27) was included in this series. The patient had terminal carcinoma of the breast with a metastatic lesion to the right cerebellopontine angle, causing hearing difficulty and vertigo. While being treated by Dr. Revici, she was investigated for the effect of lipid therapy on the symptom of vertigo. This therapy was noted to be beneficial for the symptom of vertigo but ineffective for the tinnitus or hearing difficulty.

Seven cases were eliminated because they were seen only once and no definite observations could be made. This left 51 cases. Eight of these cases presented a change in the character of the lesion, from alkaline to acid or vice versa, which was considered as representing eight additional cases, making a total of 59 cases. Two cases were eliminated because they showed no response to the substances utilized, even though they were authentic cases of peripheral vestibular cochlear disease (Ménière's disease). Although I treated them, there was no probability of obtaining a result. These two cases are discussed separately (Cases 20 and 37). This left a total of 57 cases that were analyzed for the effect of the substances developed by Revici on the symptom of vertigo and the relationship of his theory on lipids to interpretation of the character of the symptom, its evolution, and control of the disease process related to it.

Table 1 consists of the complete array of cases that were investigated. There were 51 actual cases who presented 57 abnormal foci related to peripheral vestibular disease and peripheral vestibular cochlear disease. In both these groups, the symptom of vertigo was the major complaint for which these persons sought relief.

With few exceptions, all these patients had had previous therapy with medications commonly utilized in the treatment of vertigo. Previous therapy included histamine intravenously or subcutaneously, nicotinic acid orally and by injections, hormones, salt-free diets with or without the addition of ammonium chloride, sedation with barbiturates, antihistaminics, dimenhydrinate, and allergic treatments consisting of injections of house dust and vaccine given in the belief that the vertigo was associated with primary nasal allergy. A few patients had been confined to hospitals for several weeks and had been investigated extensively in an effort to aid in obtaining relief for the symptom of veritgo. One patient was scheduled for an operation on the ear; another had a labyrinthotomy performed on one ear. These treatments failed to attain the desired result, i. e., the relief of vertigo.

TABLE 2.—*Classification According to Diagnostic Response*

Response	No. of Patients	Per Cent
Good	44	86
Unsatisfactory	5	9
None	2	5
Total	51	

There were 23 male and 28 female patients. The youngest man was 22 years of age and the eldest 73. The youngest woman was 27 years old and the eldest 67. The greatest incidence of vertigo in this series was between the ages of 30 and 60 for both men and women. In my first series of reported cases of vertigo, the greatest incidence of the symptom was between the ages of 40 and 60.

In Table 2, the patients are classified according to their response to this method of investigation. Of 51 patients, 2 showed no response, although the clinical evidence was clear-cut, indicating peripheral vestibular disease. These patients were treated for short

TABLE 3.—*Response to Treatment According to Alkaline or Acid Group*

Result	No. of Cases	Per Cent
A. Alkaline Group Treated with s-Butyl Alcohol and Other Lipobases		
Good	30	84
Unsatisfactory	6	16
Total	36	
B. Acid Group Treated with Various Sulfur Compounds, Organ Extracts, Etc.		
Good	16	76
Unsatisfactory	5	24
Total	21	

periods in order to convince myself that no response was obtained. Five patients were deemed unsatisfactory in their response to the acid and alkaline substances utilized. Treatment over short periods gave results that confirmed the initial unsatisfactory response. I could not obtain any sustained relief of the symptom. Forty-four, or 86%, of the patients gave satisfactory responses, and this conclusion was sustained by the satisfactory results of subsequent treatment.

For the purpose of clarity, the two cases showing no result are now discarded, to be discussed later in this survey. This leaves 49 actual cases; 8 of them presented a change in the character of the lesion and were successfully treated with lipid therapy, bringing the survey up to 57 investigations. The latter total appears in the following tables.

In Table 3, the cases are divided into two groups according to the alkaline or acid character of the symptom of vertigo. An alkaline pattern was presented by 36 cases, and 21 cases had an acid pattern of vertigo. A satisfactory response was shown by 84% of the cases with an alkaline pattern and by 76% of those with an acid pattern.

TABLE 4.—*Response According to Sex*

Result	No. of Cases	Per Cent
Male Patients		
Good	20	78
Unsatisfactory	6	22
Total	26	
Female Patients		
Good	26	81
Unsatisfactory	5	19
Total	31	

Table 4 shows the cases with alkaline or acid patterns of the symptom of vertigo divided according to sex. There were 26 cases in men, of which 20, or 78%, showed a satisfactory response; 31 cases were in women, 26, or 81%, showing satisfactory results. As may be noted in Table 4, there is no difference between the number of cases and the percentages of the results as tabulated according to sex.

Table 5 shows 22 cases with a diagnosis of peripheral vestibular disease, of which 16 presented an alkaline symptom and 6 an acid symptom. Thirty-five cases appeared with a diagnosis of peripheral vestibular cochlear disease, of which twenty had an alkaline symptom and fifteen had an acid symptom. In my earlier investigations on the symptom of vertigo and headache, it was evident that even in small groups of cases it is rare for like clinical symptoms to be biochemically identical. In addition, the repeated finding of biochemical dissimilarity confirms Revici's findings of the dualistic nature of the pathological foci with their associated metabolic imbalance. In my earlier papers on veritgo and head and neck pain, I have outlined his theories and their application, and I refer the reader to them. Suffice it to say that the data in Table 5 point to the futility of attempting to influence a pathological focus with one modality or substance without taking into account the fact that similar clinical phenomena, such as vertigo or headache, may be biochemically dissimilar and, therefore, the attack must be different or altered to meet the therapeutic needs of the particular situation.

TABLE 5.—*Relationship Between Acid and Alkaline Type of Vertigo and Clinical Diagnosis*

Peripheral Vestibular Disease			Peripheral Vestibular Cochlear Disease		
Symptom	No. of Cases	%	Symptom	No. of Cases	%
Alkaline	16	74	Alkaline	20	58
Acid	6	26	Acid	15	42
Total	22		Total	35	

TABLE 6.—*Results of Treatment in Fifty-Seven Cases According to Diagnosis*

Peripheral Vestibular Disease			Peripheral Vestibular Cochlear Disease		
Response	No. of Cases	%	Response	No. of Cases	%
Good	17	78	Good	29	83
Unsatisfactory	5	22	Unsatisfactory	6	17
Total	22		Total	35	

Table 6 shows the results of treatment in 57 cases according to the diagnosis. One group of 22 cases had a diagnosis of peripheral vestibular disease, and the second group of 35 cases had a diagnosis of peripheral vestibular cochlear disease. Aside from the fact that good results were obtained in both groups, 78% for peripheral vestibular disease and 83% for peripheral vestibular cochlear disease, it must be emphasized that no one substance could be used because the character of the symptom, either alkaline or acid, as illustrated in Table 5, demanded different means of attack, as was demonstrated by the substances utilized. In each one of the clinical groups in Table 6, the results obtained were predicated on the ideas of metabolic imbalance promulgated by Revici, which is thus confirmed by the clinical data submitted showing the different responses of the symptom to the broad groups of substances utilized. These substances are discussed later.

The fact that no men appeared in the peripheral vestibular disease group with an acid symptom must not be considered important. In my first series of cases in the study of vertigo, no woman appeared in the peripheral vestibular cochlear group with an acid symptom. As the series grows, it is to be expected that patients will appear in the groups which did not contain any patients previously.

A summary of my first group of cases of vertigo (published in 1953) combined with the present series of cases shows how the gaps ultimately tend to fill in. Whereas Table 7, Section B, reveals that no men with peripheral vestibular disease having an acid symptom appeared in the present series, the combination of the present series of cases with the series in my earlier article on vertigo (1953) shows that 10 cases having peripheral vestibular disease with an acid symptom come into their proper place to fill in the gap for statistical purposes. In the present series of cases, nine women appeared in the peripheral vestibular cochlear disease group with an acid symptom; in the 1953 series, no woman appeared in that category.

The cases are also classified according to sex and the alkaline or acid character of the symptom. These data are analyzed in Table 8.

Thus, it is apparent from Tables 8 and 9 that the over-all results in both series combined into one show a high percentage of cases with good outcome, 78% to 86%, whether regarded from the point of view of the character of the symptom, the clinical diagnosis, or sex. The difference of 7% seen in Table 9, Section A (Good Response), when the cases are analyzed to show the results according to the character of the

TABLE 7.—*Results in Fifty-Seven Cases of Vertigo According to Sex and the Alkaline or Acid Character of the Symptoms, 1955*

Peripheral Vestibular Disease					Peripheral Vestibular Cochlear Disease				
	Men		Women			Men		Women	
Response	No.	%	No.	%	Response	No.	%	No.	%
	A. Alkaline Group Treated with Alkaline Lipids								
Good	7	70	5	84	Good	9	90	9	90
Unsatisfactory	3	30	1	16	Unsatisfactory	1	10	1	10
Total	10		6		Total	10		10	
	B. Acid Group Treated with Acid Lipids								
Good	0	..	5	84	Good	4	67	7	88
Unsatisfactory	0	..	1	16	Unsatisfactory	2	33	2	22
Total	..		6		Total	6		9	

TABLE 8.—*Summary of Results in First Series of Forty-Two Cases (1953 Series) of Vertigo Added to Present Series of Fifty-Seven Cases of Vertigo Arranged According to Alkaline or Acid Character of the Symptom and According to Sex*

A. Alkaline Symptom (51 Cases)

	Peripheral Vestibular Disease (27 Cases)				Peripheral Vestibular Cochlear Disease (24 Cases)			
	Response: Men		Response: Women		Response: Men		Response: Women	
	Good	Unsat.	Good	Unsat.	Good	Unsat.	Good	Unsat.
1953	6	1	4	0	2	0	2	0
1955	7	3	5	1	9	1	9	1
	13 (76%)	4 (24%)	9 (90%)	1 (10%)	11 (90%)	1 (10%)	11 (90%)	1 (10%)

B. Acid Symptom (48 Cases)

	Peripheral Vestibular Disease (31 Cases)				Peripheral Vestibular Cochlear Disease (17 Cases)			
1953	9	1	13	2	0	2	0	0
1955	0	0	5	1	4	2	7	2
	9 (90%)	1 (10%)	18 (85%)	3 (15%)	4 (50%)	4 (50%)	7 (77%)	2 (23%)

TABLE 9.—*Summary of Results on Combination of Earlier Series with Present Series for Both Acid and Alkaline Symptom and Response According to Sex*

A. Series Combined for Both Acid and Alkaline Symptom (99 Cases)

	Good Response		Poor Response	
	No.	%	No.	%
Alkaline symptom	44	86	7	14
Acid symptom	38	79	10	21

B. Response According to Sex (99 Cases)

	Good Response		Unsatisfactory Response	
	No.	%	No.	%
Men, 47 cases	37	78	10	22
Women, 52 cases	45	86	7	14

TABLE 10.—*Results According to Diagnosis in Total of Ninety-Nine Cases from Both Series*

Peripheral Vestibular Disease			Peripheral Vestibular Cochlear Disease		
Response	No. of Cases	%	Response	No. of Cases	%
Good	49	84	Good	33	80
Unsatisfactory	9	16	Unsatisfactory	8	20
Total	58		Total	41	

symptom is not important, and it would be fair to say that patients presenting an active alkaline or acid symptom have an equal chance of obtaining a good result. Similarly, patients of either sex have an equal chance of obtaining a good result.

Table 10 shows that when the combined series are analyzed according to the clinical diagnosis, patients with either condition, namely, peripheral vestibular disease and peripheral vestibular cochlear disease, have an equally good chance of attaining control of the acute attack of vertigo.

In Table 8, Section B, it may be noted that a male patient having peripheral vestibular cochlear disease with an acid symptom stands a 50% chance of obtaining a good result. Had his chances for a good result been judged by the two cases that appeared in the 1953 series, both of which attained unsatisfactory results, his chances would have to be judged as poor. In the present series of cases, six men with an acid symptom appeared, four of whom obtained satisfactory results, giving a 66% chance of a good response. On combining the cases, the 50% figure is obtained.

The foregoing illustrates the desirability of continuously analzing the results for each series of cases and of combining the series so that all the cases are treated statistically as one group. From a statistical point of view, this procedure is more desirable because Revici's method of treatment is essentially the same in all cases, except that different substances with similar but more intense effects have been developed.

A hearing loss, usually of the perceptive type, was present in 32 cases, while none was noted in 17 cases of the group with peripheral vestibular disease. In no case in this series was a measurable improvement obtained after treatment was discontinued. Prior to the onset of the presenting attack, no audiometric evidence was available.

Tinnitus of varying character, such as humming, roaring, and whistling, both constant and inconstant, was present in 32 cases. In 17 cases, there was no complaint of this nature. This symptom paralleled the symptom of vertigo in intensity. When the attack was controlled, there was occasional complete cessation of tinnitus. Diminution in intensity of tinnitus was the rule when the attack was controlled. This symptom usually antedated the onset of the vertigo by varying long periods of time and was associated with the difficulty in hearing. In the recent cases, the three symptoms occurred practically simultaneously.

TABLE 11.—*Contributory Conditions Found in Fifty-One Cases of Vertigo*

Condition	Case No.	No. of Patients
Hypertension	3, 4	2
Clinical otosclerosis	6	1
Osteoarthritis	8	1
Hypochondria	9, 4	2
Cranial pressure (psychogenic)	11	1
Healed duodenal ulcer	12	1
Menopause	13	1
Migraine	14, 31	2
Chronic otitis media	16, 22, 47	3
Diabetes	17	1
Coronary disease	18	1
Radical mastoidectmy	19	1
Arteriosclerosis	7, 45	2
Bilateral antral disease	20	1
Metastatic brain tumor (breast)	27	1
Tension headache	28, 29	2
Chronic adhesive process	33, 34, 39	3
Depression (Case 34 had received shock therapy)	34, 40	2
Asthma	35	1
Cerebral palsy	36	1
Labyrinthotomy	37	1
Epistaxis	42	1
		32
No associated condition	..	19
Total		51

The symptom of vertigo was, in all cases, the disabling symptom, and the other symptoms of tinnitus and difficulty of hearing were accepted by the patient without complaint. Headache occurred in 8 cases in the peripheral vestibular group and in 21 cases of the peripheral vestibular cochlear group. Five cases in the former and seventeen cases in the latter group were relieved of headache. Usually the headache disappeared with the relief of vertigo. In these cases the patient insisted on the relief of the vertigo, which was controlled. However, in Case 3, the vertigo was controlled but there was no improvement of the headache. In Cases 9, 28, and 29, unsatisfactory results were obtained for the headache and vertigo. Cases 38 and 45 had tension headaches which were not relieved, but the vertigo was relieved.

Table 11 shows the incidence of probable contributory conditions that were found associated with the cases of vertigo. Thirty-two cases presented these associated conditions, and the cases in which they occurred are shown in Table 1 as well as in Table 11. Nineteen cases exhibited some emotional reaction, such as anxiety, fear, or annoyance.

The symptoms of nausea, vomiting, and psychological disturbances were noted in the investigation but are not enumerated because my primary interest was the symptom of vertigo. The psychological aspects of vertigo are discussed later in this article.

SUMMARY OF RESULTS OF TREATMENT

There were 51 patients who presented 57 symptoms of either an alkaline or an acid character of the vertigo. In these patients, the 57 symptoms were treated with natural lipids, such as the nonsaponifiable fraction extracted from various sources, or with *n*-butyl alcohol when the symptoms were alkaline in character. The saponifiable fractions from various sources as natural lipids, the same fatty acids with conjugated double bonds or with α-hydroxy radicals as "abnormal" fatty acids and sulfur compounds as artificial members, were utilized when the symptoms were acid in character.

In eight patients, the character of the symptom changed during the course of treatment or some time later during a recurrence. These reversals in character of the symptom were treated for statistical purposes as eight additional cases.

A. The over-all results in 57 cases showed 46, or 81%, with a good response and 11, or 19%, with an unsatisfactory response.

B. When the figures were broken down according to sex, the results were almost the same. In the male group, there were

26 cases; 20, or 78%, had a good result; 6 cases, or 22%, had an unsatisfactory response. In the female group, there were 31 cases; 26, or 81%, showed a good result, while 5, or 19%, had an unsatisfactory result.

C. There were 36 cases with an alkaline symptom; 30, or 84%, showed good results, and 6, or 16%, showed unsatisfactory results. There were 21 cases with an acid symptom; 16 cases, or 76%, had good results; 5, or 24%, showed unsatisfactory results.

D. When the cases were broken down according to the clinical diagnosis, no great difference was noted. There were 35 cases in the peripheral vestibular cochlear disease group; 29, or 83%, showed a good result; 6, or 17%, showed an unsatisfactory result. There were 22 cases in the peripheral vestibular disease group; 17, or 78%, showed a satisfactory result.

E. A combination of the cases in my 1953 series with the present series reveals that the results were substantially the same. Adding the 42 cases in my 1953 series to the 57 cases in the present series shows that, of the total of 99 cases, 82 cases, or 82%, had good results, and 17, or 17%, had an unsatisfactory outcome. The summation of the two series gives essentially the same results and better correlates the results with the effect of the substances that were administered.

COMMENT

Vertigo continues to be a challenge to the otolaryngologist and a serious handicap to the person affected with the symptom. The saitsfying help obtained with the application of Revici's methods toward the alleviation of the symptom of vertigo naturally compelled me to continue the investigation of his procedures.

In my previous article, I enumerated the various etiological factors that can affect the vestibular apparatus, constituting a stimulus sufficiently intense to initiate the attack of vertigo with complete recovery, or constituting an injury severe enough to cause the destruction of the vestibular apparatus with the cessation of the symptom of vertigo. Between these extremes, various gradations are seen.

However, when translating these multiple etiological factors to the histologic counterpart, one gets a picture of tissue fixed in death, which is termed, "degenerative, inflammatory, allergic, etc." However, the accepted picture associated with Ménière's disease is that described by Hallpike and Cairns in 1938[10] and Hallpike and Wright in 1940. The histopathological changes consist of gross dilatation of the scala media primarily,[10] with secondary involvement of the saccule and the utricle, degeneration of the sensory epithelium, and rupture of the superior semicircular canal with albuminoid coagula in the lumen. They considered that there is no evidence of the cause for endolymphatic dilatation. They suggested that the dilatation may be an effect of the disease. In its turn, the dilatation, through varying pressure changes, may be the cause of the attack.

Subsequent observers have confirmed the findings of these authors, and they have speculated in a similar fashion about the etiological factors or mechanism by which the histiologic picture is brought about. Aside from the cases of concussive trauma, fractured skull, tumors, vascular accidents, pyogenic infections, or tuberculosis of the labyrinth, all of which present a known picture, e. g., labyrinthitis or acoustic neuroma, there is resort to mechanisms of many kinds to interpret the histologic picture. In addition to the histologic picture presented by Hallpike and Cairns, other histologic data have been reported, such as cystic excrescences within the walls of the endolymphatic duct, as possible causative factors in Ménière's disease. Lempert and associates[18] presented histologic sections showing areas of vesiculation occurring along the lumen of the semicircular canals, in both the acute and the chronic forms. They theorized that toxic fluid, differing in chemical composition from normal endolymph, is released in the lumen of the endolymphatic membranous labyrinth from these ruptured vesicles. This is assumed to be the cause of the attacks of vertigo.

Guild ‡ indicated that these blebs were common findings in histologic sections. This idea is discussed by Wustrow,[13] who agrees with Guild and states that Rudinger, as early as 1864, described these patterns and considered them as normal physiological structures.

To account for these pathological findings and the symptom, other paths of speculation have been pursued. Autonomic imbalance, labyrinthitis, serous labyrinthitis, vasomotor imbalance, etc., were considered as basic mechanisms. Since no evidence of frank inflammation was present in any section, the negative finding led Wittmaack § to conclude that "hydrops of the labyrinth is in general the slightest expression of disease of the labyrinth that can be produced by an inflammatory stimulus." This plurality of mechanisms and etiologic factors deduced as theories of pathogenesis from histologic sections has given no clue to what really happens clinically in the living patient. On the other hand, no explanation is offered for cases of real Ménière's disease which are diagnosed clinically as such and which give no evidence on postmortem histologic section that any change has occurred in the vestibular or cochlear labyrinth. Arnvig,[13] Berggren ‖ and Brunner [12] report such negative histologic findings. No explanation is offered. No further purpose would be served in this discussion by citing that many theoretical comments on vertigo that are deduced from histologic considerations. The reader is referred to the excellent monograph by Williams,[14] which I have freely consulted.

Histologic findings have failed to give sufficient information concerning what actually happens at the pathological focus in the labyrinth. Administration of various substances for the relief of vertigo has likewise not provided a rational plan of action for the relief of the symptom. Many substances familiar to every physician have been tried on an empirical basis but have not consistently worked out. In any plan of therapy, one is ultimately led to the belief that while the substances utilized have had a transient but beneficial effect, eventually it was the patient's mechanism of homostasis that operated to relieve the symptom so that a remission occurred.

Revici's studies, and conclusions to be drawn from them, and their application to the symptom of vertigo, as presented in this investigation, indicate the direction in which a logical explanation can be found. The probable explanation lies in the field of biochemistry that Revici has developed in his studies on pain and cancer in the living person. It is to be sought in a biochemical imbalance that can occur spontaneously or be induced in a locality which is, for some unknown reason, predisposed to such an occurrence. This imbalance can exist for a long time without any visible or microscopic change in structure. Ultimately, structural changes may be initiated which can either go on to resolution or go into a chronic stage with destruction of the afflicted part, in this case, the labyrinth. Other changes occurring in the locality, such as vasodilation, vasoconstriction, edema, or sodium retention, are acceptable as phenomena secondary to the fundamental one of metabolic or biochemical imbalance.

In the above discussion on the histopathology of Ménière's disease, I have sought to show that no valid conclusion may be drawn from morphological considerations. They have led only to a state of confusion which is reflected in the various theories concerning the mechanism of pathogenesis and in the multiplicity of methods of treatment. Revici has pointed to the route that must be followed in order to reach the actuality of the mechanism of pathogenesis in the living. The form taken by the labyrinthine structure as seen histologically after it has been struck by a disease process has led to the general classification of the process as neoplastic, acute and chronic inflammatory, etc. The dynamics of

‡ Guild, S., in discussion on Lempert and others.[13]

§ Wittmaack, K.: Die entzündlichen Erkrankungsprozesse des Gehörorganes, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Springer-Verlag, 1926, Vol. 12, p. 290; cited by Williams.[14]

‖ Berggren, cited by Williams.[14]

the living disease process may be approached through the appreciation that many such processes express themselves through a deviation of the pH of the pericellular fluid in the immediate vicinity of the process. This concept has been evolved by Revici as one of the basic parts of the theory on metabolic imbalance in disease states. This imbalance is similar to that in diabetes mellitus, a disease which affects the general organizational metabolism and expresses itself, when uncontrolled, in the appearance of urinary glucose. In addition, the role of the "abnormal" lipids in the disease process itself led Revici to the utilization of lipids to correct that imbalance. Thus, we can arrive at a better understanding of the disease process. A ready tool is at hand for the correction of the metabolic imbalance, namely, the application of Revici's theories. By the uitilization of this method in combating the symptom of vertigo, Revici's theories have been confirmed by me at the practical level of applied therapeutics.

In his earlier studies in the field of general pathology, Revici [8] with Leroux (1941) differentiated the various morphological changes occurring in different pathological tissues. He separated these recurring cytopathological transformations into two large groups, related to the two antagonistic groups of lipids that intervene in many pathological states.

In contrast to the normal histologic appearance, the pathological cells of the lesion corresponding to the anoxybiotic type of biochemical imbalance presented the following characteristics: The cells were younger and less differentiated; the nuclei were more uniform and usually round; the chromatin and parachromatin were more diffusely spread. The cytoplasm was basophilic. The connective tissue with less differentiated cells was stained metachromatically with toludine blue. The adipose cells often had an irregular form, and the fat within the cell existed in an emulsion form in some cases. A similar picture was seen in the myeline sheaths of the nerves. The pathological conditions in anoxybiotic states were further grossly typified by vascular lesions, with proliferation of the endothelial cells of the smaller vessel walls, leading to thrombosis followed by ischemic infarcts with characteristic cellular necrosis.

On the other hand, the dysoxybiotic process in histologic sections of pathological tissue presents cells with condensed chromatin, lobulated nuclei, and oxyphylic cytoplasm. Throughout the cytoplasm, there are azurophile granules staining an intense red with methylene blue. Intracellular vacuoles are increased. Pyknosis, karyorrhexis, and karyolysis characterize most of the nuclei in their evolution with foci of cellular disintegration. As a rule, the tissues are edematous with intracellular vacuoles and accumulation of fluids, especially in the interstitial spaces. The adipose cells usually remain unchanged in the process.

Within the endolymphatic vestibular-cochlear area, the appearance of the gross histologic picture has been characterized as a hydrops. In the cochlea, the aspect is that of complete destruction with the associated changes in structure. It would be interesting if the cytological and histologic changes accompanying peripheral vestibular disease could be correlated with the systematization that Revici has described for general pathological conditions, such as wounds and tumors, and has related to the anoxybiotic or dysoxybiotic phases of metabolic imbalance of pathological foci generally. It would be also interesting if these changes could be related to the metabolic imbalance in Ménière's disease. I believe that sufficient data have been presented in this investigation that speculation in this direction is justifiable. In this connection, it is not inconsistent with Revici's theory and its application at the clinical level that a pathological metabolic imbalance may exist without any change visible with the methods of investigation available at present. It is possible that submicroscopic changes are present. Only by the opposite responses of a symptom to the administration of various groups of substances in various manners can such imbalance be assumed to exist, because of the different biochemical characteristics of these substances and their known ability to

influence the local pH in one or the other direction.

This brings the discussion to the point where a transition occurs from the microscopic level to the submicroscopic. How is one to explain cases of vertigo that go on to complete recovery without leaving any clinical trace of the attack and those cases of vertigo in which histologic evidence is entirely lacking to confirm the clinical evidence that a structural change had taken place? Cameron and Abraham cite Gavrilescu and Peters, who in 1931 ¶ introduced the term "biochemical lesion." This term was used to describe the initial change (or damage) in tissue cells which precedes any structural alteration that may be seen with the microscope. This concept embraces any abnormal biochemical processes that can take place in diseased cells. On the other hand, healthy cells would show no abnormal biochemical processes and would not present any symptom. Normal or physiological activity would prevail. This concept is entirely in keeping with Revici's theories because they envision the abnormal biochemical imbalance without or with structural alterations. Revici goes even further. Through his studies, he identifies this biochemical imbalance as existing as a dualism depending upon the type of elaborated substances excreted by the cells into the pericellular fluid as a result of these abnormal metabolic activities.

The possibility of influencing many pathological states, and particularly cancer, through synthetic liposides or natural lipids

¶ Gavrilescu, N., and Peters, R. A.: Biochem. J. **25**:2150, 1931; cited by Cameron and Abraham, in Lectures on General Pathology Delivered at the Sir William Dunn School of Pathology, University of Oxford, England, edited by H. Florey, Philadelphia, W. B. Saunders Company, 1954.

TABLE 12.—*Injectable Substances Utilized, Their Code Numbers, and the Cases in Which They Were Used*

Substance	Code No.	Case No.
Lipoalcohols		
1. Nonsaponifiable fraction of butter (cow)	LBBu 10	13, 16, 17, 19, 20, 23, 24, 25, 28, 31, 32, 34, 36, 37, 39, 40, 41, 43, 49
2. Nonsaponifiable fraction of bovine lymph nodes	LBL 1/5	6, 8, 9, 10, 11, 12, 13, 16, 21, 23, 24, 26
3. Nonsaponifiable fraction of pig intestine	LBI 10	39, 40, 41, 44, 45
4. Nonsaponifiable fraction of carp intestine	LBF 10	34, 41, 44
5. Nonsaponifiable fraction of carp intestine and 10% *n*-butanol	BBF 10	
6. Xanthophyll, 5% in oil	LBX 5	3, 5, 11, 12, 13, 21
7. *n*-Butanol, 30% (sodium benzoate)	B 31	4, 5
Lipoacids		
I. Natural Lipoacids		
A. Normal lipoacids		
1. Saponifiable fraction of placenta	LAI 5	46, 48
2. Fatty acid from carp	LAF	7, 47
B. Abnormal lipoacids		
3. Irradiated fatty acids	LAX	17
4. Conjugated fatty acids, cod liver oil	CLo	
5. Conjugated fatty acids, carp	CF 5	42, 47
6. Saponifiable fraction of pig intestine, conjugated fatty acids	CI 5	46
7. α-Hydroxy fatty acids	LH 3	14
8. α-Hydroxy fatty acids	ESO 1	33, 34, 35
9. α-Hydroxy fatty acids, butter	LHB 4	30, 50
II. Artificial Lipoacids		
10. Sulfonated oil	LSO LSO 1	1, 44
11. Conjugated fatty acids with sulfur	TCL 5	2, 7, 14, 15, 17, 18, 27, 29, 33, 34, 35, 50, 51
12. Mixture of fatty acids; transformation of molecules through selenium	SPO SPO 1	7, 30, 38, 43, 46
13. Conjugated fatty acids and sulfur	SGF 5	7, 17, 38, 43, 45
14. α-Hydroxy unsaturated fatty acids with sulfur	LHSS	14, 30, 33
15. Methyl thioglycolate	MTG 5	17

led me to administer them in cases of vertigo in order to attempt to neutralize the effect of the "abnormal" lipids occurring in these states. These chemical agents, having the solubility characteristics of lipids when administered systemically, will be selectively taken up by the tissues in which a lipid imbalance exists. That the administration of proper lipid substances corrected the imbalance and relieved the majority of the cases of the symptom of vertigo offers a logical explanation for their action in affording relief from the symptom and confirms Revici's theories. It is not difficult here to pursue the argument further that similar reasoning would account for the beneficial effects in a purely "biochemical lesion" with submicroscopic changes as defined above.

An infinite number of metabolic processes are involved in the physiological activity of the normal cell and in the abnormal activity of the pathological cell. Revici's investigation led him to the conclusion that "abnormal" lipid imbalances are especially important in the pathogenesis of pathological foci. Very early in his work, Revici realized the importance of the role that these "abnormal" lipids play in determining the two different types of metabolic processes that occur in pathological tissue. These "abnormal" lipids are responsible for the elaboration and accumulation of acid or alkaline compounds.

In the present state of knowledge, no adequate explanation is possible for the fact that peripheral vestibular disease, with or without cochlear diseases, remains confined or localized to the structures of the inner ear, the endolymphatic vestibular cochlear apparatus. At most, the total volume of tissue involved is about 0.12 cc.

The idea of a biochemical imbalance with or without structural changes leads to the concept of the limiting value of boundaries. Without this concept, no localization is possible, and it is conceivable that once a pathological focus started it would ultimately destroy the total organization. While this unfortunate end-result is realized all too frequently when cancer of an area is considered, it is happily not the case in vestibular cochlear disease, the pathological abnormality now under discussion.

The normal lipids are factors that play an important role in determining the formation

TABLE 13.—*Dualistic Patterns Observed in Cancer and Pathological States Applied to the Pathogenesis of Labyrinthine Disease (From Revici*)*

	Predominance of Sterols	Predominance of Fatty Acids
Cellular changes	Marked proliferation and nondifferentiation of cells	Increased rate of aging and death of cells; intracellular vacuolation; hydrops of cells
Metabolic changes (causing pain or vertigo)	Local acidosis	Local alkalosis; edema
Vascular changes	Thrombosis	Lysis of walls of organ

* Revici, E.: Control of Cancer with Lipids, presented at Clinical Pathological Conference, Beth David Hospital, New York, May 9, 1955.

TABLE 14.—*Levels of Involvement with Pathogenesis As Applied to Disease of Labyrinth (Vertigo) (Modified from Revici)*

Anatomical Level	Physiopathological Process	Clinical Phase
Nuclear	Pathogenesis; original changes	Chronic condition
Intracellular	Abnormal metabolic activity; sterol-fatty-acid imbalance	Active condition spread to involve labyrinth; homeostasis cannot reverse process
Intercellular	Liberation of acid or alkaline substances	Attacks of alkaline or acid headache, tinnitus, deafness, vertigo
Blood vessels	Vascular changes in labyrinth	Vascular headache, intensification of symptoms, lesions in organ
Blood and other organs	Systemic metabolic changes in organ or labyrinth, especially indirect through vomiting, etc.	Ultimate death of labyrinth, several symptoms or no symptoms

of the separating boundaries. Revici's theories embrace the ideas of holism or organicism (Smuts,[19] Needham and Emerson #), the basis of which is that the whole is more than the sum of its parts (de Robertis [20]). This principle implies different levels of organization to include the entire organism but may be applied to the study of cellular function. The cell can be considered, thus, as an entity in itself.

Following Revici's concepts, the organization of the several organs can be seen as a function of progressive barriers. Starting from the nucleus of the cell, we may progressively go to the cell, the pericellular fluid, the lymphatics, the vascular components, then to the organs, and finally to the entire organism. Invasion or progress of a pathological process may be limited by one barrier or may involve further levels and be destined to attain a broader volume of localization. Beginning in one cell, it may spread to tissue, then to an organ, and finally to the entire organism. In peripheral vestibular cochlear disease, the entire cochlear organ and part of the vestibular organ is ultimately destroyed.

Each of the levels presents its own unique characteristic manifestations. Thus, intracellular changes lead to anabolic changes associated with processes of growth. Catabolic or dysoxybiotic processes, through various gradations, may ultimately lead to the death of the cell. Intercellular fluid alterations lead to changes in the intercellular pH due to the elaborated acid or alkaline substances and, therefore, to alkaline or acid symptomatology. Organic or structural formations produce manifestations related to the specific function of the organ. Generalized changes are recognized through systemic changes and the effect on the various organs.

These barriers are realized through the intervention of the lipids. Formed by a polar group bonded to a predominantly nonpolar group, the lipids are attracted toward the watery solution in which only the polar group enters. The predominantly nonpolar group prevents the complete entrance or solubility in the watery solution. The result is an orientation of these lipids forming molecular layers at the surface of the watery solution or protoplasm. Thus, a boundary membrane is realized.

The aliphatic constitution of fatty acids makes for a perpendicular disposition of these molecules toward the aqueous surface. Since these molecules are not bound together, they will permit the easy passage of other molecules between them. The permeability is relatively high. Lipids with a polycyclic nonpolar group also form molecular layers, but the nonpolar group will orient itself parallel to the surface. The sterols will form impermeable layers. These two types of formations make small areas or spots in the membranes of biological entities, and their relationship will affect the amount of permeability of the membranes. Other factors, e. g., calcium and metals, which bond the fatty acids at their polar groups will also decrease the permeability. Any substances that bonds the polar group of fatty acids will have this effect.

Besides the above role, fatty acids when unsaturated intervene in an oxidation metabolism, transforming the inactive molecular oxygen into an active atomic form. Neutralization of an active normal unsaturated fatty acid acts through its effects on membrane permeability and the metabolic activity of the acid.

Abnormalities in the constitution of the fatty acids, such as conjugation of the double bonds or the appearance of the fixation of radicals at the double bonds or at the second carbon, induce abnormal processes which characterize the pathological foci. These biochemical changes involving the changes in the structure of the fatty acids convert them into abnormal types which the organism is incapable of reversing. This creates an imbalance in the sterol-fatty-acid content of the cell. It is at this stage that a lesion can be related to chronicity because of the incapacity of the organism to change that imbalance.

Needham and Emerson, cited by de Robertis and others.[20]

The relationship of the pathological focus to the general metabolic changes of the entire organism is a close one in many conditions. The body is organized into entities. Barriers separate these entities from one another. Two systems related to the maintenance of homeostasis are of fundamental importance in maintaining the relationship between the body and the pathological focus. One system concerns the hydrosoluble constituents of the body; the other concerns the liposoluble constituents of the body, i. e., fatty acids and sterols. The hydrosoluble system is characterized by changes that are rapid in their evolution. The liposoluble system is characterized by changes that are slow in their evolution. As I pointed out in my communication on head and neck pain, they are time reactions.

In the application of the above concepts of the organization to labyrinthine disease, the labyrinth may be considered as an entity with its own boundaries and its own pathological relationship to the total organization. The lesion in labyrinthine disease being small, about 0.12 cc. in volume, it is theoretically unlikely that it would influence the general body metabolism. No clinical or laboratory evidence would substantiate that idea. On the other hand, the general metabolism could influence the intensity of activity of the lesion in the labyrinth. This is confirmed by the fact that the symptom of alkaline vertigo, for example, is more intense at night, when the body is more alkaline, or immediately after eating, because the body is usually alkaline at that time. In this example, the local lesion is influenced by the hydrosoluble system with its characteristic rapidity of action, as mediated through the acid-base balance of the body. However, the effect of lipidic mobilization can also be rapid, but only if it is mediated through the hydrosoluble system. This is an indirect action. The administration of acidifying or alkalinizing substances induces neutralization or increases the hydrosoluble imbalance with limited effects. The administration of lipids has a much more intense effect which lasts much longer.

The above considerations become clear when the intervention of the body defense mechanisms is utilized during periods of stress. The hydrosoluble adrenal secretions act rapidly, but the effects are of short duration. The corticosteroids are liposoluble, and their effects are more profound and of longer duration. Other steroids that are produced by the body seemingly operate in a similar fashion. This point of view concurs with Revici's theories of biochemical imbalance and can explain the influence exerted by other conditions, such as metabolic processes, acute or chronic infection at a distance from the pathological processes, and food intake. Indirectly, the pituitary and other glands of internal secretion play a part.

Repeated emotional states of fear, anger, rage, or annoyance, associated with current conscious situational problems or elicited by disease processes such as vertigo—persistently recurring emotional states that are associated with highly personalized unconscious stress situations, often connected with long past psychic trauma—create the biochemical environment that can influence the pathological focus. In contrast to these states, there are also purely psychogenic factors which are really subconscious mechanisms associated with the general problem of hysteria. These situations are imitative and not real. They have no correlation with organic lesions. The projection of the symptom, i. e., vertigo, headache, etc., to an organ or a locality cannot be referred back to some physiological process, anatomical site, or actual pathological change. Under observation for a period of time, these patients do not react to the opposing substances that are utilized to determine whether the character of the symptom is acid or alkaline. Even when a real biochemical lesion exists and the symptom can be shown to react to opposite-acting substances, the psychogenic or hysteroid factor or overlay, i. e., depression, hypochondria, is not influenced. My observations on vertigo confirm this view, which is entirely consistent with Revici's theory. At no time is the patient aware of

what the substance is that is administered, and certainly he is unaware of how its effect on the metabolic imbalance will influence the symptom.

On the other hand, the psychosomatic factors, whether initiated by the activity of the lesion itself, i. e., fear because of unknown implication of the symptom of vertigo, or by some transient or recurring life situation causing fear or anger, can influence the intensity of the symptom of vertigo. In this instance, it is not the projection of the psychic factor to a locality that is important; here the patient has no choice. The labyrinthine symptom of vertigo may be provoked or intensified if the pathological focus is active. This point of view based on Revici's studies helps the clinician to better understand the interplay of psychological and pathological factors when treating vestibular disease, headache, etc. This discussion in no way underestimates the importance or the desirability of psychiatric treatment. Such treatment is not possible or even desirable in every case. The objective is to relieve the headache or vertigo when this can be done, so that other modalities may be utilized to help the patient. On the other hand, the psychosomatic factors may be continually operative so that no treatment is successful. Case 37 of this series, reported earlier in this article, illustrates this aspect.

Relationship of Vertigo to Other Factors, Such as Psychological Factors or Foci of Infection.—The relationship of the small abnormal focus occurring in peripheral vestibular cochlear disease, expressed as a localized metabolic imbalance, to other large active foci at a distance from the inner ear is interesting. For many years, clinicians have noted the relationship between the vertiginous attack and active gall-bladder disease, recurrent attacks of appendicitis, and acute exacerbations of chronic pelvic disease. Other foci of disease, such as asthma, infected teeth, and acute attacks of tonsillitis, have been in similar relationship. I have discussed this phase in my previous papers, and such cases occur frequently enough to cause concern regarding the outcome of any method utilized in the treatment of vertigo. Revici's method is no exception when a similar difficulty arises. However, there is this difference. Understanding of this complicating factor was limited to the rather vague statement that circulating toxins or humors derived from the distant focus adversely affected the abnormal vestibular or vestibular cochlear focus, so that, despite treatment, the symptom could not be controlled. It was not apparent from the clinical point of view that an abscessed tooth or chronic catarrhal gall-bladder disease, for example, affected the total organization of the person. These foci affected the tooth or gall-bladder region only. But it was easily determined that, through Revici's methods, the administration of *n*-butanol for an alkaline active lesion of the gall bladder could initiate or intensify an attack of vertigo predicated on a vestibular lesion that was acid in character.

For instance, asthma is an alkaline symptom which was provoked in treating a backache with an acid symptom. This situation has fortunately not occurred frequently. In this instance, the patient had been confined to bed because of a severe backache. I was not aware that she also had asthmatic attacks. By administering sodium thiosulfate solution every three hours, I relieved her backache so that she could get out of bed, but the continued administration of sodium thiosulfate solution provoked a moderately severe asthmatic attack. I had recourse to epinephrine inhalations and an occasional injection to control the asthmatic attacks until the backache was sufficiently relieved.

There is no need to invoke toxins, humors, or other obscure mechanisms to account for this interplay of symptoms. The situation is sufficiently understandable, through Revici's researches, on a more fundamental level; namely, the metabolic imbalance in the abnormal focus and the intervention of "abnormal" lipids and the elaborated substances produced by the activity of the lipid imbalance in the pericellular fluids associated with the abnormal focus. It is clear, then, that one focus may influence the metabolism of another focus in a manner similar to that

of the administration of the lipid predominant in the abnormal focus.

In the same way, emotional states can influence the intensity or provoke an attack of vertigo. This factor has been noted in my earlier article. Pathological structure changes, as noted in the preceding paragraphs, influence the abnormal vestibular focus through their liberation of hydrosoluble and "abnormal" lipid substances which are the products of an abnormal metabolism; these substances act via the blood stream and can be at quite a distance from the vestibular focus. On the other hand, emotional states, such as fear, anger, rage, or anxiety, are part of a normal physiological process related to the general problem of homeostasis. In turn, such states are associated with the liberation of epinephrine and arterenol which are hydrosoluble and, according to Revici's theories, would act much like $NaHCO_3$ in their influence on the abnormal focus. This would be their nonspecific effect. They would also have a beneficial effect on an acid symptom and intensify an alkaline symptom through their action on the elaborated substances of the abnormal focus concentrated in the pericellular fluid of the lesion. The corticosteroids probably would also be liberated, but later. The steroids, according to the theory, would act on the cellular level through the liposoluble system and, in this way, diminish the intensity of an alkaline symptom.

It would be premature to state which substance produced in emotional tension states contributes to the initiation of the symptom of vertigo. However, recent work as described by Funkenstein [9] from the Harvard Medical School and by von Euler [21] indicated that arterenol and epinephrine secretions may be specific indicators which distinguished one emotion from another. These authors showed that anger directed outward is associated with short periods of increased arterenol, and that anxiety or anger turned inward, i. e., depressed states, is associated with long periods of increased epinephrine formation. The physiological response would be an increase in blood pressure, the specific hormonal effect of epinephrine or arterenol. The nonspecific effect would be the intensifying action on an alkaline symptom. These ideas are purely speculative from this point on, for one must also account for the kinds of corticosteroids liberated in these emotional states.

The above discussion shows the manner in which emotional states may influence the symptom of vertigo. It is not reasonable to suppose that a vestibular lesion about 0.12 cc. in volume can, by its activity, influence the entire metabolism of the individual patient except through the nausea, vomiting, and increased perspiration that take place during an attack of vertigo. These are purely physiological effects. It has been observed that chronic recurring periods of fear and anger can influence the intensity of the symptom of vertigo in many instances through the above mechanism. This fits in well with Revici's hypothesis and its practical clinical application in the treatment of the symptom of vertigo. The evidence presented in this investigation sustains the ideas here discussed.

The liberation of corticosteroids is an endeavor of the body to counteract the effect of the fatty acids, and particularly of their "abnormal" members such as conjugated fatty acids, which intervene in the pathogenesis of abnormal foci. The "abnormal" fatty acids appear, according to Revici's hypothesis, as active lipids inducing permeability of membranes and metabolic changes in the cells. The mobilization of the steroids acts as an antagonist, reducing the permeability of cell membranes and also the metabolic activity of the fatty acids.

Again, I wish to emphasize that the chronicity of a condition is related to two factors. One is the incapacity of the organism to overcome the predominance of the "abnormal" lipids. The other factor is the capacity of the pathological focus to produce or create large amounts of "abnormal" lipids characterizing the lesion. The operation of these factors may also explain the tendency

of the lesion to progress and to involve in the abnormal process not only new and similar entities (cell to cell, cochlea to utricle, utricle to semicircular canals, etc.) but also higher levels of organization, namely, the entire peripheral vestibular cochlear apparatus.

It is to be understood that endolymphatic hydrops with vestibular involvement may remain in the alkaline or acid phase of the metabolic imbalance for long periods with no change in the character of the imbalance. This phase of the investigation of vertigo was confirmed in this series. In eight cases, the lesion changed its character of metabolic imbalance from alkaline to acid, or vice versa. Similar observations were reported in my previous communication on headache.

In Ménière's disease or endolymphatic hydrops, the process starts in the cochlea, then extends further and ultimately invades the vestibular labyrinth. It must be assumed that the barriers beyond this organ are potent enough that the process is inhibited from further extension. The ultimate destiny of the diseased labyrinth is its conversion to inactive scar tissue, the least active of tissues. This terminates the disease but at the expense of the total destruction of the organ.

Some of my failures with the clinical application of Revici's hypothesis may be attributed to actual insufficiency of the method as utilized. Other failures may be more directly attributed to the mechanism of drug resistance.* The body opposes the intervention of any medication which by itself creates a certain state of metabolic imbalance. The study of the effects of lipoacids has ultimately led to the search for and the use of synthetic liposides. Revici has furnished me with many lipids for administration to patients with the symptom of vertigo, having in mind the possibility that a more specific action would be realized from some of them. This effect is difficult to evaluate at this time. Even if such specificity were present in a preparation, its nonspecific character would prevail.

*Reference 17. Revici, E., and Taskier, F.: Antitoxic Effects of Adrenals Upon Abnormal Fatty Acids, to be published.

Operative Indications.—In this investigation, I have been constantly aware of the admonition that operative intervention in patients having peripheral vestibular cochlear disease with impaired hearing beyond the serviceable level should not be advised until all medical methods have been exhausted in the effort to arrest the symptom of vertigo. Nevertheless, in this series, patients have presented themselves for whom established operative procedures were advised for the relief of the symptom of vertigo. In these, fortunately, I have been gratified to have obtained good results with Revici's method so that no operation was necessary. All patients were told that if this method failed, and other accepted methods also failed, only then would I consider operative intervention. Almost all the cases presented in this study had been treated unsuccessfully by the accepted methods of therapy for vestibular disease; they were able to obtain relief from the attacks and their recurrent attacks were also controlled by means of lipid therapy. About 20% of the cases had an unsatisfactory outcome, and they may have sought relief in operative intervention elsewhere, but I have no record of such occurrences.

Use of Revici's Method as a Therapeutic Approach.—In view of the good results presented in this study, I believe this method of therapeutic approach to the problem of the relief of vertigo should attract the serious attention of otolaryngologists, so that, with its application to the problem on the part of many workers in the field of otolaryngology, Revici's method may be integrated with accepted methods of current therapy for the relief of the symptom of vertigo. In this manner, knowledge of the disease in the living can be extended.

The confirmation of my results by others would also add a practical means of investigation and therapeutics to present methods, and where a good result is attainable, unnecessary operative intervention could be avoided.

The accepted therapy has been restriction of fluids and sodium chloride, with the

substitution of NH_4Cl for the salt, and administration of histamine intravenously and subcutaneously and of KCl, 6 to 10 gm. in aqueous solution by mouth daily. In addition, dimenhydrinate, antihistamines, nicotinic acid, ergot derivatives, atropine, etc., have been utilized, and last but not least in popularity have been vitamins, on the theory that a subclinical avitaminosis was present. Sedation with barbiturates has been used fairly frequently and appears to be on the increase. The theoretical considerations prompting these methods of therapy either are based on empiricism alone or were derived from the consideration of many pathogenic theories. To say the least, the treatment of vertigo has become very difficult and confusing. Revici reduced the attack to a fundamental level of biochemical metabolic imbalance on the basis of his studies which are applicable to general pathological conditions. Their application to the problem of the treatment of vertigo by me in this presentation indicates that, from the practical point of view, their utilization in the field of applied therapeutics in otolaryngology is very desirable. They fill a need for a systematized understanding of the pathological process and, at the same time, furnish a method and means to attack the problem.

In all my discussions on the method used in this investigation, the word "treatment" must be defined in the sense that it is being here utilized. The word is used from an investigational viewpoint. The understanding is that "treatment" is used to show that a correlation exists statistically between the administration of a substance and a favorable response. Should Revici's and my findings be confirmed and the method of therapy generally accepted and utilized, the word "treatment" would have its usual meaning.

The associated conditions of varying degrees of impaired hearing that accompanied the symptom of vertigo in a substantial percentage of the cases also came under scrutiny in this and my previous investigation. The fact that the impaired hearing antedated the onset of vertigo in the cases in which these two symptoms were associated led to the conclusion that the two conditions were part of the same pathogenic process but separated in time. It would be just as logical to assume that the symptom of vertigo and the impaired hearing were the end-result of two different pathological mechanisms. I have not observed any improvement in the cochlear defect but have persistently seen the beneficial effects on the symptom of vertigo with the application of Revici's method. On the other hand, I have not observed a case of vertigo in which impaired hearing did ultimately develop. It would seem that the question of an effective barrier of biochemical nature could be raised in this connection as a tentative theory explaining this difference. I have never observed a labyrinth that did not respond to caloric stimulation after the attack of vertigo had subsided in a case in which there was no hearing loss. Further study on this point in conjunction with Revici's method is indicated.

It was hoped not only that the symptom of vertigo would be controlled but that the pathological focus in which the fatty-acid-sterol imbalance was present would be restored to a normal ratio. Unfortunately, this was not the case. The acute attacks of vertigo were controlled in the majority of cases, and recurrences were also successfully controlled. Many of the cases that had had frequent recurrent attacks prior to the treatment with lipids have been followed, and no recurrences have been observed. However, the conclusion cannot be drawn from the study and research thus far that the metabolic imbalance had been restored to normal or that structural alterations, where at all reversible, were reversed to the normal healthy state. The only warranted conclusion from this study is that a large group of substances, having different chemical structures and solubility characteristics and related to the general class of lipids, are capable of alleviating the symptom of vertigo on the basis of metabolic imbalance as proposed by Revici. The fortuitous result of no further recurrence in the elapsed period of time since the therapy was instituted will need further study in

order to evaluate that aspect of the problem. It is again emphasized that a permanent arrest or cure of the symptom is not to be concluded from this study.

I believe that sufficient clinical data have been presented in this communication to show that the symptom of vertigo may be controlled by the use of many substances which could be classified under the generic term "liposides." This word was coined by Revici to designate those substances having polar-nonpolar groups with characteristic solubilities. They are so designated because they include not only the naturally occurring substances usually thought of as lipids but also a large group of other synthetic substances having similar activities.

The above considerations alone would be sufficient reason for presenting this investigation. However, not only are we confronted with a tool for use in relieving vertigo, but we have at our disposal an entirely new means of systematizing knowledge in the field of otolaryngology, which is additional reason for this presentation.

Beginning as I did with the idea of holism or organicism as a framework, I have indicated the pathway which we as otolaryngologists should take in speculating on the theories associated with the problems in our specialty. We can direct our thinking along such paths, at least provisionally, so that we can account for the clinical facts associated with the problems of pain and vertigo that I have now and previously presented. Unfortunately, there are no data on the pathological cytology of the cochlear or vestibular organ that can be directly correlated with the metabolic lipid balance manifest in cases of active vestibular disease. But, because of the different response of the symptom of vertigo to different groups of substances, it may be at least tentatively inferred that a metabolic imbalance does exist in the injured cells of the cochlear duct and of the semicircular canals and that it is not necessarily associated with a structural change.

As with all theories that have a practical application at the clinical levels, modifications will take place from time to time as further research on the part of other observers indicates that such changes are necessary. The presentation of the data in this communication indicates that Revici has given us a method of systematizing otolaryngological data not only at the basic laboratory level but also at the clinical level. Revici's proposal of such a large group of substances that may be utilized for so many different conditions, i. e., pain, vertigo, itching, asthma, and cancer, should not surprise us because the same physiological and pathological, biochemical, and metabolic mechanisms have been with us ever since life was present on this planet.

SUMMARY AND CONCLUSIONS

A series of 106 cases with the symptom of vertigo has been presented in this and a previous paper and analyzed according to Revici's concept of a metabolic imbalance which exists in pathological foci and manifests itself as a local alkalosis or acidosis.

Seven cases were eliminated for lack of data. This left a total of 99 case studies. This figure represents the sum of 42 cases termed the 1953 series and the present series of 57 cases. The two series were combined and analyzed according to sex, alkaline or acid character of the symptom, and clinical diagnosis.

The analysis showed an average good result of 80% for both series. This percentage indicates a consistent correlation of the use of Revici's method and substances with the results achieved. Additional substances from various sources were utilized in the present study. It is here again emphasized that I have submitted this article for the practical reason that the method described is simple and has been successful in its application. In addition, it gives a new systematization to clinical data in the field of otolaryngology. Finally, this communication points to a vast group of substances from widely differing sources and of different structures which have similar biochemical activities. The substances presented here are but a few, but they open a pathway for the development of other substances having simi-

lar biochemical activities. It is to be hoped that these, too, may have a practical application in the field of otolaryngology.

Views on cell permeability and their relationship to fatty acids and sterols are added.

The role of foci of infection and psychological aspects in their relationship to the symptom of vertigo is amplified.

The simplicity of this method of therapy is emphasized, and its integration with a new method of systematization of clinical knowledge is indicated.

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The Problem in Endoscopic Topical Anesthesia

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THE ISSUE

When I was a medical student, I was made aware that from time to time a patient unexpectedly died as a result of cocaine hydrochloride anesthesia. It was my impression at the time that this was one of the calculated risks of topical anesthesia. Sollmann [1] reports a death occurring from use of 0.2 cc. of 10% cocaine. Holinger states that a death resulted from administration of 2 cc. of 10% cocaine. As a resident, I witnessed the accidental infiltration of 10 cc. of 10% cocaine in a nasal plastic procedure without incident. A confrere stated recently that he pays no attention to dosage; he aims toward anesthesia, watching carefully for signs of reaction, and so far has not had a death. In 1948, I surveyed the membership of the American Broncho-Esophagological Association, and below are the ranges of dosage for cocaine and tetracaine (Pontocaine) hydrochloride in the more commonly used strengths:

	Cocaine 10%	Tetracaine 2%
Preliminary pharyngeal spraying	2 bursts to 5 cc.	0.5 cc. to 5 cc.
Preliminary applications to piriform fossae	2 drops to 4 cc.	0.5 cc. to 10 cc.
Intratracheal instillation (one physician used up to 5 cc. of 20% cocaine)	0.5 cc. to 5 cc.	0.5 cc. to 10 cc.

The range of dosage is wide, and, in spite of what one may feel about the upper limits given, the implication is that no one experienced an undue number of severe reactions or deaths. It is also worth noting that the cocaine users are more cautious than the tetracaine users. More will be said about this later. It might seem on first glance that those who hold that dosage is not a prime factor in the problem of toxic reactions have a point.

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From the Veterans Administration Center.

HOW SERIOUS IS THE PROBLEM?

Seevers,[2] a pharmacologist of Ann Arbor, Mich., in a paper presented before a symposium on anesthesia at the 1948 meeting of the American Academy of Ophthalmology and Otolaryngology, stated that ". . . there are probably more deaths from local anesthetics in this country than from any other single class of compounds in common use." He asked for a show of hands from those who had witnessed a toxic reaction or a death from topical anesthesia during the past five years and estimated that 30% to 40% of the audience responded. Seevers also stated that the use of barbiturates in ordinary dosage in the hope of preventing toxicity

> has led to a situation in which the incidence of deaths from local anesthetics in this country is appalling. Unfortunately, most of these serious reactions are ascribed to individual sensitivity or idiosyncrasy to the drug rather than to overdosage, and a large fraction of such cases do not appear in the clinical literature.

Idiosyncrasy will be discussed later also.

In 1948, the Veterans Administration requested a group of consultants to prepare a bulletin [3] on the subject of topical anesthesia. In a personal communication from a member of the group, it was stated: "A significant number of deaths were occurring throughout the country."

In 1952, a letter was issued from the office of the Chief Medical Director of the Veterans Administration, which stated:

> Reports of the continued used of excessive amounts of cocaine and pontocaine as topical anesthetics with resultant deaths are being received in this office.

As Seevers pointed out, no over-all figures are available for the country as a whole for topical endoscopic anesthesia per se.

Beecher and Todd [4] made a study of deaths associated with anesthesia in 10 large university hospitals from 1948-1952 and reported on their findings in 599,548 anesthetizations of all types. The over-all death rate was a surprising 1 in 1560 cases. The authors remarked on the reaction of surprise of the participants in the survey at their own figures, showing the inaccuracy of clinical impression as opposed to the carefully recorded facts. The only finding of note reported on topical anesthesia was that the use of tetracaine dropped by one-half over the four-year period of the survey. Unfortunately, the information on endoscopic topical anesthesia was limited, as no records were kept on anesthetizations performed by endoscopists. The implication is that the death rate is considerably higher for the country as a whole than available figures indicate.

In addition to the above, the following deductions also seem valid and applicable to the problem. In the experience of any one physician, regardless of the drug or amount of drug used, severe reactions or deaths are not common. The human body has a wide range of tolerance to topical anesthetic agents, which belies the findings in animal experimentation, largely based on parenteral methods of testing. Regardless of the paucity of unfortunate experiences in the history of the individual physician, the combined experience has brought to the attention of the profession, to some extent, the dangers inherent in cocaine. Some of this secondhand caution has gone by the board as regards tetracaine, and this is reflected even in the larger clinics, according to Ireland's survey of approximately 40,000 cases [5]:

	Ratio of Cocaine to Tetracaine
Incidence of drug use	4.3:1
All toxic reactions	1.2:1
Severe reactions and deaths only	1.0:2.2

One article states that the incidence of reactions to tetracaine is 1 in 100 cases without barbiturates and 1 in 500 cases with barbiturates. The same author in a later article reported a death from 25 cc. of 1% tetracaine used as a gargle in preparation for an esophagoscopy, and the opinion rendered was that the patient was sensitive to tetracaine; there was no consideration of the possibility of overdosage.

OTHER ASPECTS OF THE PROBLEM

The problem also concerns other aspects of topical anesthesia. How did it come to pass that tetracaine, a more toxic, more locally irritating, and more antigenic drug, achieved such popularity? What has technique, spray or application to the piriform fossae, to do with the problem? What about barbiturates, idiosyncrasy, concentrated or dilute solutions, epinephrine, etc.? In terms of the ideal anesthetic agent, what are the shortcomings of cocaine? It is an efficient agent, relatively nonirritant and nonallergenic. The major shortcoming is its toxicity. Any substitute worthy of supplanting it must, first of all, be less toxic. Tetracaine is six times more toxic, according to Rubin's [6] work, in which agents were applied topically on animal mucosal surfaces. In addition, it is more irritating and produces a higher incidence of allergy [7]: a ratio of 18:1 as against cocaine. Hexylcaine hydrochloride and lidocaine hydrochloride, on the other hand, legitimately bear promise of supplanting cocaine after a thorough clinical trial. In 1930, Knoefel * used intravenous cocaine on cats, with the suggestion that death was due to direct paralysis of the heart. Less attention appears to have been paid to his simultaneous use of subcutaneous cocaine, with the suggestion that death was respiratory. One year later, Fussgänger and Schaumann, working in the I. G. Farben Laboratories, developed tetracaine. In their summary, they reported that all animal deaths from toxic doses of tetracaine were respiratory, thus implying that resuscitation by means of artificial respiration was possible. However, in the body of their paper they exhibited tables showing control studies

* Knoefel, cited by Shumacker.[8]

with other agents, including cocaine, and all animal deaths from cocaine were also respiratory. The timing and the implication appear to have borne fruit. In my paper in 1949,[10] this subject was discussed more fully, especially the implication from the literature that what are described by some authors as the separate convulsive and collapse types of reaction, with respiratory death in the first and cardiac death in the second, are actually the same process, the only difference being the speed with which a lethal dose is built up (Schumacker [8]). Some time ago, a nationally known anesthesiologist wrote me that tetracaine and cocaine should not be used at one session as the patient would then be in double jeopardy; the cocaine could affect the heart and the tetracaine the respiration. In his opinion at that time, tetracaine was obviously the drug of choice. In a recent publication he no longer makes a choice between the two drugs but issues a warning that when treating a convulsive reaction due to tetracaine with intravenous thiopental (Pentothal) sodium it would be wise not to withdraw the needle too soon, as the convulsive seizures may recur owing to the comparatively slow rate of detoxification of tetracaine in the body. A recent article stated that when reactions occur following tetracaine the prompt administration of thiopental sodium intravenously, artificial respiration, and oxygen inhalation have proved so effective that no death following tetracaine occurred in the past 1000 cases. Is the author saying in effect that he is still encountering reactions but by heroic means has avoided a death? Would it be presumptuous to suggest that heeding the manufacturer's warning as to dosage control in the first place might have led to a report of no reactions requiring heroic measures?

Several authorities have condemned without qualification the use of topical anesthetic agents by the spray technique. Would it not be more appropriate and in keeping with established facts that the injudicious use of drugs, by whatever technique, spray or application to the piriform fossae, be condemned? Rubin,[9] in testing animals under topical conditions on several mucosal surfaces, found that the total dosage of the drug in milligrams was the chief factor in determining the severity of toxic reactions. The tolerated dosage could be increased by slowing the application of the drug.

In the symposium on topical anesthesia at the 1955 meeting of the American Broncho-Esophagological Association, one of the panel members asked the guest anesthesiologist, Dr. Stage, whether he felt dosage was a factor in the toxic reaction. Dr. Stage replied that he would have to go along with dosage and the time taken in applying that dose. By either technique, severe reaction or death ensues when a near-lethal or lethal dose is built up faster than detoxification mechanisms can dissipate it.

To illustrate further the futility of the controversy over technique, I shall refer to a published article anonymously. The authors state that, after some unfortunate experiences, including a death, they ran a series of 1000 cases. A resuscitation team was set up for the management of toxic reactions. Because the authors apparently had been using the spray technique and felt they were not adequately able to estimate the dosage, a change was made to the piriform fossae application. A total of 10 cc. of 2% tetracaine was placed in a medicine glass and 1 cc. reserved for intralaryngeal instillation. The remaining 9 cc. was dipped into with piriform applicators, and two applications of one minute each were made bilaterally. By gravimetric methods they estimated that an average of 1.5 to 2 cc. of drug was used. They experienced 19 reactions, 7 of them severe. They were unable to explain this higher incidence of reaction, as they termed it. Rubin,[9] as already stated, found the relative toxicity of tetracaine to cocaine in animals to be 6:1; in other words, toxicity-wise, 1% tetracaine is equivalent to 6% cocaine. The toxicity equivalent of the 10 cc. of 2% tetracaine used was that of 20 cc. of 6% cocaine or 20 cc. of 1% tetracaine. Carabelli [11] reported on 621 cases in which he used the equivalent of 1.0 cc.

of 2% tetracaine, diluted to 8 cc. of 0.25% tetracaine, and obtained good anesthesia without incident. Carabelli stated:

> Review of the literature shows that all authors committing themselves on the actual quantity of pontocaine used for bronchoscopy or bronchography are using far more of the drug than is recommended by the manufacturer.

The manufacturer states: "The total absorbed dose of Pontocaine hydrochloride should not exceed 20 mgm." This maximum safe dose is 1 cc. of 2% tetracaine or its diluted equivalent. Clark, Orkin, and Rovenstine [12] report that the duration of action of 0.5% tetracaine is twice that of the 0.25% solution, and that with 1% solution the duration of anesthesia is only 25% longer, and they feel that this does not warrant the increased risk of toxicity. Obviously, dogmatically changing the technique alone is not the answer.

I should like now to discuss idiosyncrasy or, as some term it, individual sensitivity. Goodman and Gilman [13] distinguish between idiosyncrasy and hypersensitivity and state they do not have the same connotation. These authors define idiosyncrasy as an abnormal or unusual response to a drug manifested by a pharmacodynamic action which either is greater than would be anticipated or is of an unusual pattern. The example given of an unusual pattern is the response of excitation rather than depression to morphine. As an example of a pharmacodynamic action greater than anticipated, they cite cinchonism in response to a small dose of quinine.

Hypersensitivity, on the other hand, is an allergic reaction to a drug. Penicillin can be given in a relatively unlimited dose without causing toxicity, while in a hypersensitive person a small dose may elicit a violent reaction. The severe hypersensitivity reaction is likened to anaphylactic shock, with smooth muscle spasm and increased capillary permeability the characteristic pathological findings in the shock organs affected. While it is true that urticaria and bronchial spasm may not be evidenced in severe cases as diagnostic leads, and that rapid circulatory collapse with or without convulsions may make distinction from the toxic death difficult, necropsy may give a lead. Urbach and Gottlieb [14] cite the following necropsy findings in anaphylactic deaths from various sources in the literature: (1) pronounced inflation of the lungs with marked bronchial narrowing, not relaxing even in death, (2) abnormal numbers of eosinophilic leucocytes in the bronchial walls, (3) occasional cerebral and laryngeal edema attributable to increased capillary dilatation, (4) necrotizing fibrinoid arteritis of smaller vessels, and (5) a hyperplastic reticuloendothelial system. When none of these findings (urticaria, bronchospasm, necrotizing arteritis, etc.) are manifested in a topical anesthesia death, and the dosage is not in the microscopic range of idiosyncrasy, one cannot lightly disregard the possibility of toxic reaction and hide behind the terms idiosyncrasy, sensitivity, or hypersensitivity.

Returning to idiosyncrasy, Goodman and Gilman state that it occurs rarely and bears no relation to dosage and that little is known about its mechanism. Certainly, no one would question the classification of the death reported by Sollmann with 0.2 cc. of 10% cocaine as idiosyncrasy. I have already referred to Seevers' statement with regard to the unfortunate ascribing of severe reactions and deaths to idiosyncrasy rather than to the actual culprit, overdosage. Is there an arbitrary dose below which a reaction can be labeled idiosyncrasy and above which it can be labeled overdosage? This is obviously impossible from our present knowledge of the normal range of tolerance. I feel that a death occurring from 2 cc. of 10% cocaine would represent an unusually low tolerance, bordering on idiosyncrasy. By the same token, 10 cc. of 10% cocaine infiltrated into the tissues without reaction would represent an unusually high tolerance. Among the procedures listed in the literature for routine total dosages below the equivalent of 2 cc. of 10% cocaine are those of Carabelli [11] for tetracaine and those of King and Jones [15] and my own [10] for cocaine. I

know there are others not recorded in the literature.

There has also been opinion expressed with regard to the relative merits of concentrated and dilute solutions. Sollmann [1] has pointed out that when longer duration of anesthesia is desired more concentrated solutions are indicated; dilution, he states, slows absorption of cocaine. Rubin [6] found that dilution in itself did not reduce toxicity. As he suggests, the answer lies in the different situations of testing. Where testing is done from a subcutaneous pool, thus with a limited surface available for absorption, dilution would be a factor in slowing absorption, while from a relatively unlimited mucosal surface there would be little difference. However, dilution does play a definite role of slowing absorption if the agent is fractionally applied, drop by drop, as it takes a longer time then to apply the same drug dose when it is given in a larger quantity of solution. It is for this reason that I feel that there is definite advantage in using 2 cc. of 5% cocaine as against 1 cc. of 10% cocaine for intratracheal instillation (besides, there is less drug base wasted through initial coughing). By the same token, it is better to use 2 cc. of 2% cocaine than its equivalent, 0.4 cc. of 10% cocaine. This brings up a point of major import. While an expert, experienced operator can make a small amount of solution go a long way, the less adept will frequently find the smaller amount of solution expended before adequate anesthesia is attained and so he will be tempted to use more solution. If he is handling concentrated solutions or a drug like tetracaine, he may find himself working near the upper limit of tolerance of many patients and he will eventually experience a number of toxic reactions; depending on chance, he may strike one or more patients with low tolerance limits, and thus severe reactions or deaths will ensue. As Adriani [16] and Seevers [3] have cautioned, "Use the least amount of the least toxic drug," and to this I would add, "fractionally applied in the most dilute solution compatible with efficient anesthesia." It may be that the techniques of Carabelli, King and Jones, and myself, separately arrived at but developed with the same aim, have approached the maximum of safety compatible with efficient anesthesia.

I should like at this point to relate my experience with more than 5000 cases. In 1949, I reported three allergic reactions: two proved as due to tetracaine and one probably due to tetracaine. I also reported four toxic reactions: one death and three mild reactions. All four toxic reactions occurred within a period of three months, shortly after the series was begun in 1946. At that time, the technique used was an approximate 2 cc. of 2% tetracaine spray followed by the intratracheal instillation of 1 cc. of 10% cocaine. After the series of reactions, the quantity of spray was accurately measured, and it was found that for the more difficult patients as much as 4 cc. of 2% tetracaine spray was used. From then on the tetracaine spray was used more cautiously. I found I was able to get aditus relaxation with 1 to 2 cc. of tetracaine solution, but the residents were having difficulty. I then changed to 2% cocaine spray and later changed from 1 cc. of 10% cocaine intratracheal instillation to 2 cc. of 5% cocaine. Application to the piriform fossae was tried in 299 cases, but as I familiarized myself with the literature I became convinced that total drug dosage was the answer, and my aim was to attain the minimum efficient drug dosage by whatever technique. I have now recorded more than 2000 cases in which 2% cocaine spray and 5% cocaine instillation were used, with only a single mild reaction. This patient became faint and developed a tachycardia as his only manifestation. This technique represents an equivalent of 1.4 cc. of 10% cocaine or 0.16 gm. of drug base. By dilution this dosage is delivered in 4 cc. of solution. When 4 cc. of 2% spray occasionally becomes necessary, the total dose becomes 0.2 gm.

Whether or not to use epinephrine has also caused some confusion. Sollmann,[1] Gurd and Sachs,[17] Lierle,[21] and the Mayer report of 1924 [18] have covered the synergistic action of cocaine and epinephrine, suggesting that the use of epinephrine with cocaine is un-

wise. Epinephrine is more rationally used with tetracaine. Fussgänger and Schaumann [9] and Rubin [6] found a marked protective effect when the combination was tested in animals subcutaneously. Rubin found, however, that epinephrine actually increased the toxicity for tetracaine when applied topically to the bronchial mucosa, varying with the speed of administration. Goodman and Gilman [18] state that epinephrine reactions are not uncommon when this drug is given with local anesthetic agents.

NEWER DRUGS

Since 1940 three new drugs have appeared. Propoxycaine (Pravocaine or Ravocaine) hychochloride can be dismissed, as its toxicity is similar to that of tetracaine. Hexylcaine (Cyclaine) and lidocaine (Xylocaine) bear definite promise. Crawford [19] feels that lidocaine is superior to hexylcaine in all respects except duration of anesthesia. Lidocaine is recommended in 2% strength for topical anesthesia and hexylcaine in 5% solution. Hexylcaine is reported equivalent to cocaine in the same strength as to potency but is one-half as toxic, on the basis of animal studies only so far. Two schools of dosage appear to have sprung up. Rovenstine † feels that 8 to 10 cc. of 5% hexylcaine provided adequate anesthesia for bronchoscopy in a series of more than 1000 cases. A report on 58 cases by Ray and Vinson [20] mentions using 20 cc. of 5% hexylcaine; this is the manufacturer's recommendation in a brochure. The first group uses the toxicity equivalent of 2.0 to 2.5 cc. of 10% cocaine. The second group uses the toxicity equivalent of 5 cc. of 10% cocaine. The latter dose may not prove to be innocuous over the long run. Experience with thousands of cases alone will tell the story.

SUMMARY

In the experience of any one physician, the number of severe reactions and deaths is not large. Some protection is afforded in the fact that most patients have a wide range of tolerance to topical anesthetic agents. The evidence, indirect as it may be, indicates an unduly high mortality rate for the country as a whole. Few physicians experience many deaths, but many experience a few deaths. When cocaine was the only commonly used drug, the combined experience of the entire profession led to a degree of caution, which appears to have fallen by the board with regard to tetracaine (Pontocaine) hydrochloride. Tetracaine achieved its popularity through an unfortunate misinterpretation of its attributes, which appeared to represent it as an improvement over cocaine.

The evidence, exclusive of the rare idiosyncrasy, points to drug dosage and the time employed in applying that dose as the prime factors in the toxic reaction. The reliance on barbiturates and epinephrine sets up false gods. While the expert may attain safety employing small amounts of solution, the average physician can best protect himself by giving himself leeway through dilution of his drug dose in a relatively large quantity of solution. A safe maxim would be: Administer slowly the least amount of the least toxic drug in the most dilute solution compatible with efficient anesthesia. The newer drugs, lidocaine and hexylcaine, offer great promise, but do not alter the maxim, and extensive clinical trial will be required to determine their true place.

† Rovenstine, E. A., from the narration of the film, "Local Anesthesia with Cyclaine in Hospital Practice," 1954.

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Audiology Section

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Thresholds of Overload in Normal and Pathological Ears

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Studies in the physiology of hearing have revealed a most significant fact, that is, that the aural harmonics studied so long by psychologists as one of the peculiar attributes of the hearing process actually arise in the hair cells of the organ of Corti.[1] The importance of this is that by using the pyschologist's techniques for detecting and measuring these subjective tones the range of action of the hair cells themselves can be determined.

The type of distortion that produces harmonics is nonlinear distortion. This occurs when a driven system fails to respond with proportional increases to further increases in amplitude of the driving force. In the ear the first structures to overload in this manner are those of the organ of Corti. The structures of the middle ear and the fluids of the inner ear respond in a remarkably linear fashion up to an amplitude of sound vibration that would have already destroyed the sensory structures lying along the basilar membrane.

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As a pure tone is raised in intensity from some low level the sensory structures respond with proportional increases. When the electrical potentials arising from the action of the hair cells are measured their voltage output doubles when the amplitude of the sound is doubled. This effect continues until the amplitude of the forcing vibration becomes too much for the hair cells and, as any mechanical system behaves under similar conditions, overloading occurs and nonlinear distortion with its concomitant harmonics of the driving fundamental results. These harmonics (overtones) can be recorded in the electrical responses of the ear and can be heard by the experienced observer in his own ear. It has been suggested [2] that in human ears the sensation level at which this overloading occurs is a measure of the dynamic range * of the ear and reflects the physiological condition of the hair cells at the time the measurement is made. The level at which distortion first becomes audiometrically evident we have called the threshold of aural overload.

Lawrence and Blanchard [2] suggested the use of this measure in normal ears for the detection of those that might most easily be damaged by excessively loud sounds. The evidence was brought forth that distortion and trauma occur at the same anatomical locus, the hair cells, and that it is the amplitude of sound vibration that is responsible for both of these effects. The overload

* This term, dynamic range, is variously defined. Here we refer to the range of response from minimum to a level that would be injurious. Peak dynamic range usually refers to the range of response of a system from minimum to maximum regardless of the distortion introduced or, in this case, the injury produced.

threshold in a number of subjects with normal audiometric thresholds was measured and found to vary over a considerable range. They made the interpretation that those with the shortest range, i. e., those that overloaded at the lowest sensation level, would be most susceptible to damage by loud sounds, and in a small number of animals showed that the ears most readily damaged were those that took the least amount of sound (above 700 cps) to overload the ear.

On the basis of these observations it is natural to investigate the influence that known inner-ear pathology has on the overload threshold. However, before interpretations can be made from the results on ears with any form of pathology present, it must be determined whether middle-ear lesions alter the remarkably linear behavior of this conductive mechanism.

The study presented here has, therefore, been undertaken with a threefold purpose:

1. To verify the range of overload thresholds as reported by Lawrence and Blanchard by making the measurements in a larger number of normal ears.

2. To determine the effect of middle-ear pathology on overload threshold.

3. To determine the effect of inner-ear pathology on overload threshold.

MEASURING THE THRESHOLD OF AURAL OVERLOAD

In the untrained ear subjective harmonics arising in response to a pure-tone stimulus can best be detected by use of an exploring tone which beats with the harmonic when the frequencies and intensities of the two are close together. With use of this method, the beginning of nonlinear distortion is revealed by finding the lowest intensity level of a tone at which its first overtone (second harmonic) can just be heard. A detailed description of the technique, precautions taken, and calibration of equipment are given by Lawrence and Blanchard; briefly, the technique is as follows:

In order to provide a fundamental and an exploring tone for the aural harmonic, two oscillators (General Radio Type 1304-A), each followed by an output voltmeter and attenuator, are fed to a matched mixing and attenuator circuit, which in turn connects through a switching arrangement to two sets of high-fidelity binaural earphones with ear cushions. With this arrangement the experimenter can test either ear of the subject and listen at the same time. Figure 1 shows a block diagram of the apparatus. A separate switch provides for the presentation of either oscillator alone or of both together through the mixer. Since in testing pathological ears it is sometimes necessary to deal with a patient with one ear considerably better than the other, means for switching in a white noise masker are provided. To assure that aural harmonics only are being measured, the second harmonic from the oscillator must be at least 60 db. below the acoustic level of the fundamental.

The output of each oscillator is also fed into the horizontal and vertical channels, respectively, of a cathode ray oscilloscope, which is used for setting the frequency of the exploring tone oscillator near that of the harmonic so that beats will be produced.

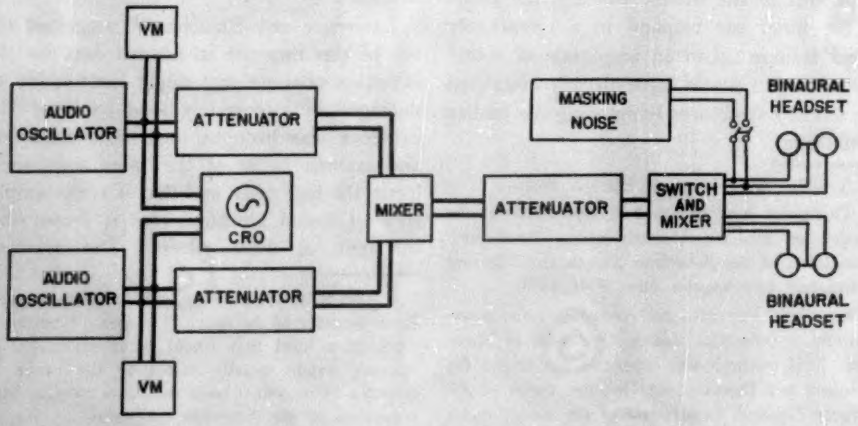


Fig. 1.—Arrangement of apparatus for measuring the threshold of aural overload.

This is done by adjusting the frequency of the exploring tone until the Lissajous figure, which is in a two-to-one pattern, is motionless. Then by means of the cycle increment dial on the oscillator the frequency is altered by 4 cps, which in a preliminary experiment seemed to give the best beats.

After thresholds for these two frequencies (the fundamental and exploring tone) are taken, the fundamental frequency is set so that when presented it will be 70 db. above the subject's threshold for that frequency. The exploring tone is made to differ from the second harmonic of the fundamental by 4 cps and then adjusted so as to be 60 db. above the subject's threshold for that tone. The tones are then switched to the subject's ear under test, and in most cases he will hear beats, with only a slight adjustment of the exploring tone level being necessary for him to hear best beats. If beats are not heard the level of both tones is raised until beats are readily perceptible. By means of the common attenuator following the mixer both tones are then reduced together until the subject claims the beats are no longer heard. A slight reduction in the exploring tone generally makes the beats return, and the level of both is again reduced with the common attenuator. This procedure is followed until the beats have disappeared completely. At this point the intensity level of the fundamental is recorded and considered to be the threshold of aural overload.†

All the measurements have been made at fundamental frequencies of 1000 and 2000 cps because these fall within the range of important speech frequencies and because the first series of experiments indicated that use of the higher frequencies reveals more accurately than use of the low an unhealthy condition in the inner ear.

The testing in the following series was carried out in a quiet, sound-insulated room.

All participating subjects received a pure-tone audiometric examination. An ear was considered normal if the frequencies used as fundamentals in the distortion test and 4000 cps (the frequency used as the second harmonic of 2000 cps) were heard by air or bone conduction within ±10 db. of the zero line on the audiogram. Ears falling below the +10 db. level were considered abnormal and a thorough classification as to whether the loss was of a conductive or perceptive type made by further otological and audiometric examinations.

† For the more recent measures in this series an overload tester manufactured by the Maico Company, Inc., was used. The inherent distortion characteristics of this instrument are even less than those of the laboratory equipment described.

RESULTS ‡

I. Thresholds of Aural Overloads in Normal Ears

Forty-six male and 31 female subjects meeting the requirements of normality were administered the threshold of aural overload test. The ages of these subjects ranged from 17 to 36 years; 52 of the 77 subjects were between the ages of 17 and 20.

The histograms in Figure 2 show the results of these tests. One hundred forty-five ears were tested at 1000 cps and 133 ears at 2000 cps. The mean level above each subject's threshold at which a tone begins to show nonlinearity is 57 db. for 2000 cps and 52 db. for 1000 cps.

The spread of the individual differences becomes greater at the higher frequencies, which is reflected in the size of the standard deviation. At a fundamental of 2000 cps this is 17 db., and at 1000 cps it is 13 db.

A comparison of these results with those of Lawrence and Blanchard shows the means obtained here to be 13 db. lower at 1000 cps and 8 db. lower at 2000 cps than those found in the earlier report. The standard deviations, however, have remained within 1 db. of the earlier value, which verifies the rather extensive range of overload thresholds, as indicated in the original report. These thresholds, as shown by the present data, vary from 5 to 80 db. above the subject's threshold at 1000 cps and 5 to 90 db. at 2000 cps. The lowering of the means has probably been brought about by refinements in the technique of detecting the overload threshold.

II. Thresholds of Aural Overload in Abnormal Ears

The abnormal ears reported here are those of patients of the Outpatient Clinic in the University Hospital. On the basis of a thorough otological examination the individual ears of these patients were categorized as to the type of lesion responsible for

‡ The majority of the results presented here were collected by Dr. Yantis as part of a doctoral dissertation in the Department of Speech, University of Michigan.

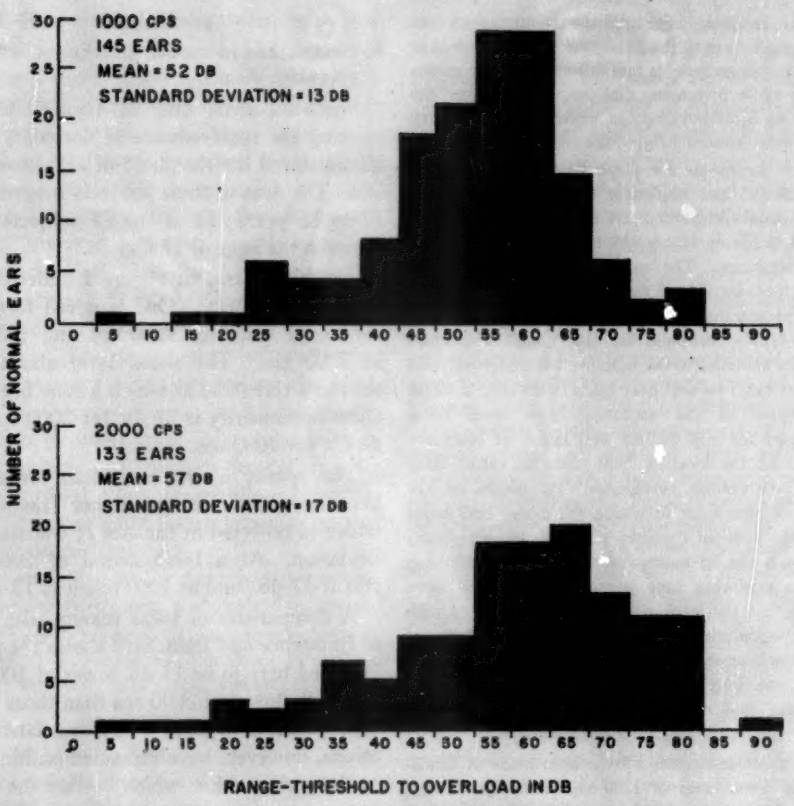


Fig. 2.—The variation of overload thresholds among normal ears.

the hearing loss, and for the purposes of this study each ear has been considered as a separate entity. Alternate monaural and binaural loudness balance recruitment tests were performed when possible, since this procedure has been shown to be fairly conclusive for determining inner-ear lesions.[8]

The results of the tests on these abnormal ears are presented in Table 1. As indicated in the column headings, the right or left ear is first designated, then the results of the recruitment tests. The types of recruitment tests employed here are not, of course, always possible to administer, but where results were obtained they have been recorded as additional information on the individual case. Indicated in the fifth and ninth columns is the amount of hearing loss in decibels as measured on the audiometer at the particular frequencies. The 6th and 10th contain the level in decibels above the audiometric zero which the tone has to be raised to produce the first audible overtone as recognized by the exploring tone technique used in the overload test. The 7th and 11th columns tabulate the difference between the two preceding columns. This has been headed "range" and is the level in decibels above the person's own threshold at which overloading occurs. This, of course, does not represent the maximum sound the person can stand or even the most comfortable loudness. It is an objective measure indicating the linear range of operation of the sensory cells, and it is this range that is considered significant.

Six patients were clinically diagnosed as having pure conductive type losses in one or both ears with pathology confined to structures of the middle ear. Five of these

TABLE 1.—*Data Obtained by Aural Overload Test and Recruitment Tests in Thirty-Five Patients with Abnormal Hearing*

Case No.	Pathology	Ear	Decibels at 1000 Cps: Recruitment	Decibels at 1000 Cps: Loss at Threshold	Decibels at 1000 Cps: Level at Overload	Decibels at 1000 Cps: Range: Threshold to Overload	Decibels at 2000 Cps: Recruitment	Decibels at 2000 Cps: Loss at Threshold	Decibels at 2000 Cps: Level at Overload	Decibels at 2000 Cps: Range: Threshold to Overload
	Middle Ear									
1	Otosclerosis	rt	NT	25	75	50	NT	25	85	60
2	Scar tissue; chronic otitis	rt	—	35	95	60	—	50	110	60
3	Otosclerosis	rt	—	45	95	50	—	35	80	45
		lt	—	45	90	45	—	50	105	55
4	Otosclerosis	rt	NT	35	85	50	..	..	..	..
5	Otosclerosis	lt	..	..	..	..	NT	60	120	60
6	Otosclerosis	rt	..	..	..	..	NT	45	95	50
	Cochlea									
7	Ménière's disease	lt	+	65	75	10	+	65	85	20
8	Neomycin toxicity	rt	..	..	..	..	P	55	80	25
9	Acoustic trauma	lt	..	..	..	..	+	35	60	25
10	Ménière's disease	rt	NT	65	75	10	NT	65	70	5
		lt	NT	55	65	10	NT	55	70	15
11	Ménière's disease	rt	P	65	90	25	NT	75	100	25
12	Acoustic trauma	rt	+	60	75	15	+	75	85	10
13	High fever	rt	+	40	55	15	+	55	65	10
		lt	+	35	55	20	+	45	70	25
14	Ménière's disease	lt	+	45	55	10	+	40	55	15
15	Ménière's disease	lt	+	20	45	25	+	25	60	35
16	Presbycusis	lt	..	..	..	..	P	75	105	30
17	Acoustic trauma	lt	..	..	..	..	P	55	80	25
18	Fractured cochlea	rt	+	80	80	0	+	80	80	0
19	Ménière's disease	rt	+	50	70	20	+	60	60	0
20	Ménière's disease	lt	+	65	75	10	+	55	75	20
21	Ménière's disease	lt	..	..	..	..	+	40	60	20
22	Ménière's disease	lt	+	60	60	0	+	70	75	5
23	Ménière's disease	lt	NT	55	65	10	NT	45	60	15
24	Ménière's disease	rt	NT	40	55	15	..	..	..	..
		lt	NT	35	50	15	NT	40	60	20
25	Acoustic trauma	rt	..	..	..	..	NT	45	60	15
		lt	..	..	..	..	NT	50	85	35
26	Ménière's disease	lt	+	80	80	0	+	70	80	10
	Eighth Nerve									
17	Acoustic neuroma	rt	P	70	95+	25+	..	..	..	..
27	Cerebellopontine angle tumor	rt	..	..	..	..	NT	20	85	65
		lt	..	..	..	..	NT	25	90	65
28	Cerebellopontine angle tumor	rt	NT	15	55	40	NT	15	55	40
29	Cerebellopontine angle tumor	rt	—	85	105	20	..	..	..	..
		lt	—	35	70	35	—	20	70	50
	Questionable Locus of Lesion									
1	Otosclerosis (mixed loss)	lt	P	55	85	30	NT	30	80	50
4	Otosclerosis (mixed loss)	rt	..	..	..	..	NT	35	95	60
		lt	NT	50	90	40	NT	45	95	50
16	Presbycusis	rt	NT	60	85	25	NT	60	90	30
		lt	NT	60	90	30	..	..	..	..
30	High fever	lt	—	80	105+	25+	—	45	90	45
31	Congenital loss	rt	NT	50	60	10	NT	70	85	15
		lt	NT	60	70	10	NT	60	85	25
32	Presbycusis	rt	NT	15	65	50	NT	25	90	65
		lt	NT	20	70	50	NT	35	85	50
33	Presbycusis	lt	NT	50	70	20	NT	55	80	25
34	Otosclerosis (mixed loss)	rt	NT	40	70	30	NT	50	70	20
		lt	NT	50	85	35	NT	50	95	45
35	Otosclerosis (mixed loss)	rt	NT	45	85	40	NT	40	90	50
		lt	NT	60	90	30	NT	50	90	40

The abbreviations are as follows: +, recruitment present; —, recruitment absent; P, partial recruitment present; NT, no recruitment test possible.

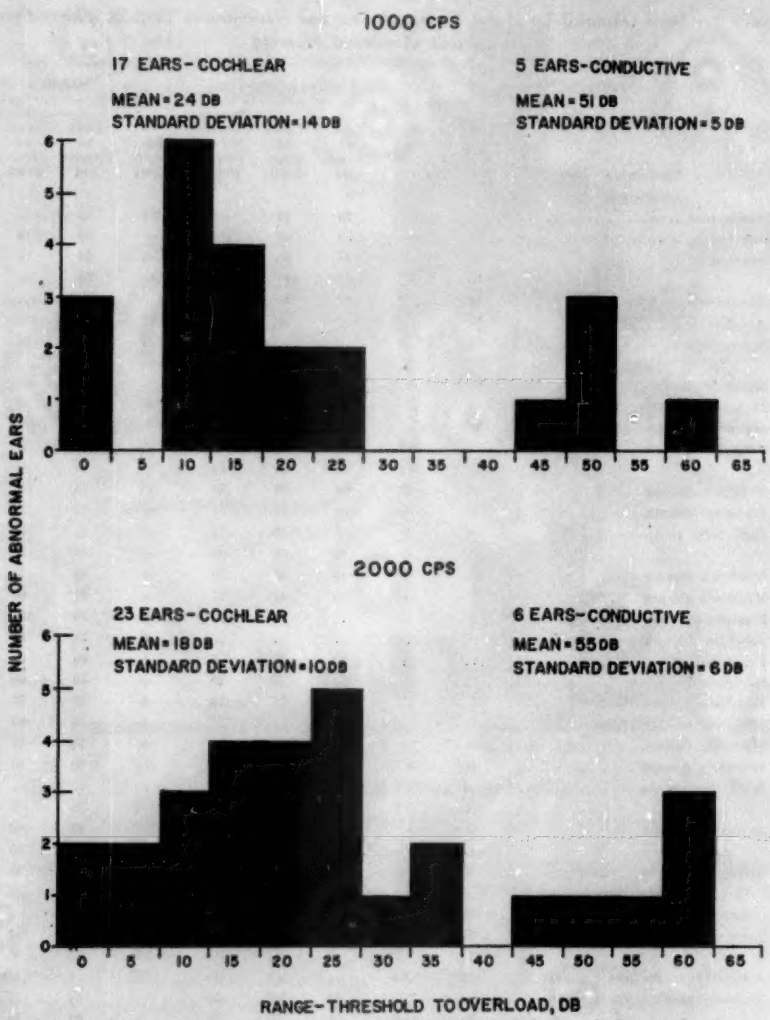

1000 CPS
17 EARS - COCHLEAR
MEAN = 24 DB
STANDARD DEVIATION = 14 DB
5 EARS - CONDUCTIVE
MEAN = 51 DB
STANDARD DEVIATION = 5 DB
2000 CPS
23 EARS - COCHLEAR
MEAN = 18 DB
STANDARD DEVIATION = 10 DB
6 EARS - CONDUCTIVE
MEAN = 55 DB
STANDARD DEVIATION = 6 DB
NUMBER OF ABNORMAL EARS
0 1 2 3 4 5 6
0 5 10 15 20 25 30 35 40 45 50 55 60 65
RANGE - THRESHOLD TO OVERLOAD, DB

Fig. 3.—The variation of overload thresholds among a series of abnormal ears.

were cases of otosclerosis and one a long-standing chronic otitis.

Twenty patients had in one or both ears for one or both of the frequencies used in the aural overload test a hearing loss due to inner-ear pathology. This was determined by an otological diagnosis and/or the presence of loudness recruitment in those patients with whom it was possible to employ the test. In most of these cases either Ménière's disease or acoustic trauma was involved. There is little doubt but that these were inner-ear lesions. Presbycusis, however, has and has not been so classified. This, no doubt, is because there are apparently different causes of this old-age deafness. In those cases where there was no evidence of recruitment, the cause of the deafness may well lie outside the cochlea, but a very important factor contributing to many cases of presbycusis is a progressive deterioration of the end-organ which starts at the extreme basal end of the cochlea.[4] In Case 16 recorded in Table 1 this may have been so; therefore, on the basis of the fact that there was partial recruitment at 2000 cps in the

left ear, these data have been included in the inner-ear category.

The data from the conductive loss cases and inner-ear loss cases have been gathered together and recorded in the histogram plot of Figure 3. The figures along the abscissa are the levels above the individual thresholds at which overloading occurred. This also is an expression, then, of the range as defined here: from threshold to overload. There is no overlap in these two series, and their means are widely separated.

A comparison with Figure 2 reveals that the mean range for the conductive loss cases is within 2 db. of the means for the normals. The standard deviations are less in the abnormal group than in the normal, but there are considerably fewer cases, which might account for this.

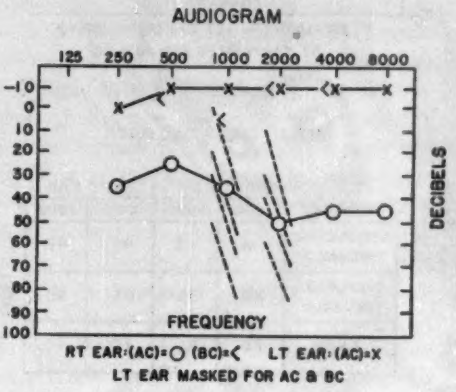
The mean range for the group with inner-ear pathology is extremely low, some ears showing the presence of aural harmonics at the audibility threshold. For 1000 cps the mean range is 24 db. which is 28 db., or more than two standard deviations, below the mean of the normal group. For 2000 cps the mean is 18 db. which is 39 db. lower than the normal group mean. This is 5 db. more than two standard deviations from the normal.

There is quite a bit more variability at 1000 cps with a standard deviation of 14 db. than at 2000 cps, where there is a standard deviation of 10 db.

Referring again to Table 1, a third category includes four patients diagnosed as having a hearing loss due to the involvement of the eighth nerve. These could not all be tested thoroughly because of the peculiar characteristics of the sound as perceived by the listener.

Little can be said about the eighth-nerve cases except that the range from threshold to point of overload is generally higher than that of the ears with cochlear involvement. It is difficult to interpret just what takes place when a normal inner ear overloads at a normal level but is followed by a nerve incapable of relaying the information. This is a subject for further investigation.

A fourth category has been added because of the possible significance the overload test might have in further clarifying the diagnosis. There are nine of these included under the heading "questionable locus of lesion." Among these there are four that would generally be classified as deafness of the mixed type, with both air and bone conduction thresholds elevated but to an unequal degree. The bone conduction threshold is generally



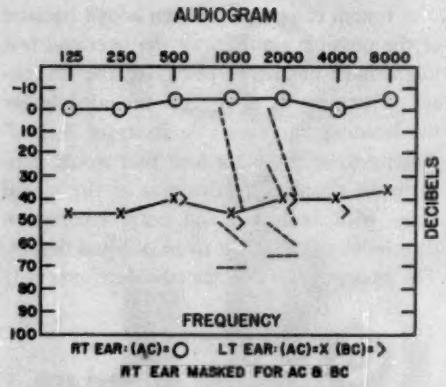
AURAL OVERLOAD DATA

	RIGHT EAR		LEFT EAR	
FREQUENCY	1000	2000	1000	2000
AUDIOMETRIC THRESHOLD	35	50	-10	-10
OVERLOAD THRESHOLD	95	110	55	50
RANGE	60	60	65	60

DECIBELS

Fig. 4.—Audiometric data from a patient (Case 2) with a pure unilateral conductive loss. The range of hearing from threshold to overload is normal.

better but not sufficiently so to indicate a normal middle ear. There are others in this list in which it was typically difficult to localize any one locus responsible for the deafness. Because of the generalized loss it is not possible to carry out the conventional recruitment tests, but the overload threshold results may have considerable significance in determining the involvement of the hair cells. Because this is such a heterogenous group



AURAL OVERLOAD DATA

FREQUENCY	RIGHT EAR 1000	RIGHT EAR 2000	LEFT EAR 1000	LEFT EAR 2000
AUDIOMETRIC THRESHOLD	-5	-5	45	40
OVERLOAD THRESHOLD	70	55	55	55
RANGE	75	60	10	15

DECIBELS

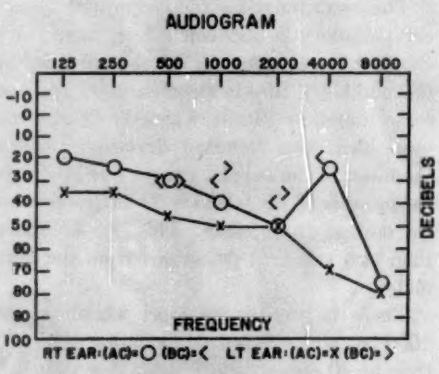
Fig. 5.—Audiometric data from a patient (Case 14) with a pure unilateral perceptive loss due to inner-ear pathology. The range of hearing from threshold to overload in the left ear is extremely limited.

and the results are so variable no means have been calculated.

In order to clarify the audiometric data from which the above information has been gathered, three illustrative cases have been selected: one from the conductive loss group, one from the inner-ear group, and one from the questionable group. The first two have been selected because each patient had one normal ear, which served as a control for comparison with the normal mean of overload threshold.

Figure 4 presents the audiometric data from Case 2. This was a young man 18 years of age who had chronic otitis in his right ear for 10 years, which finally resolved itself in the formation of considerable scar tissue and adhesions. His left ear was normal. The dashed lines represent the results of the right ear recruitment test, which was negative. The conductive loss in the right ear was 35 db. at 1000 cps and 50 db. at 2000 cps. The overload threshold occurred at 95 and 110 db., respectively, giving a range of 60 db. This is slightly above the mean for the normal group but the same as that for his normal left ear. The range from threshold obviously has not been changed by the middle-ear pathology; it has only shifted to a higher intensity level.

Figure 5 presents the audiometric data from Case 14. The patient was a 34-year-old woman with all the symptoms of Ménière's disease and with deafness and recruitment in the left ear. The audiometric threshold for 1000 cps is elevated 45 db. and that for 2000 cps, 40 db. The dashed lines show the positive results of the alter-



AURAL OVERLOAD DATA

FREQUENCY	RIGHT EAR 1000	RIGHT EAR 2000	LEFT EAR 1000	LEFT EAR 2000
AUDIOMETRIC THRESHOLD	40	50	50	50
OVERLOAD THRESHOLD	70	70	85	95
RANGE	30	20	35	45

DECIBELS

Fig. 6.—Audiometric data from a patient (Case 34) with a bilateral mixed deafness. The range of hearing from threshold to overload is less than normal but not as restricted as in the ear with extensive inner-ear pathology. The range is greatest in the left ear, in which the bone conduction indicates better cochlear function.

nate binaural loudness balance recruitment test. The right ear was normal. At 1000 cps the sound need be raised only 10 db. above threshold in the affected ear for it to overload and produce the audible harmonics. At 2000 cps this range is 15 db. The amount of hearing loss in this patient is comparable to that in the patient with the conductive loss, but the levels of overload are considerably different. In both cases the normal ear had an overload level equal to or higher than the normal group mean.

Figure 6 presents the audiometric data from Case 34, classified as mixed deafness. The patient was a 64-year-old woman with long-standing otosclerosis. Both ears show an involvement of the inner ear, which is not necessarily related to the otosclerosis. In the right ear at 1000 and 2000 cps the bone conduction is only 10 db. above the air conduction curve, which shows a loss at these frequencies of 40 and 50 db., respectively. The level of overload is 30 and 20 db. above their respective audibility thresholds. These values are slightly higher than the mean of those recorded in the inner-ear loss group but lower than those of the normal or conductive loss ears. The patient's left ear shows this effect more markedly. At 1000 cps bone conduction is 25 db. better than air conduction, which shows a loss of 50 db., and at 2000 cps bone conduction is 15 db. better than the air conduction threshold, which is also at 50 db. The overload levels are 35 and 45 db., respectively, which are midway between the means of overload for conductive loss cases (also normal ears) and inner-ear loss cases. This suggests a possible quantitative relationship between range from threshold to overload and kind of pathology causing the loss, another subject for further investigation.

TABLE 2.—*Means and Standard Deviations in the Normal, Middle-Ear, and Inner-Ear Groups*

	1000 Cps		2000 Cps	
	Mean (Db.)	Standard Deviation (Db.)	Mean (Db.)	Standard Deviation (Db.)
Range: Threshold to Overload				
Normal ears.....	52	17	57	13
Middle-ear pathology	51	5	55	6
Inner-ear pathology..	24	14	18	10
Overload Thresholds: Above Audiometric Zero				
Normal ears.....	49	13.5	55	14
Middle-ear pathology	88	7	99	14
Inner-ear pathology..	69	13	73	12

COMMENT

Table 2 summarizes the means and standard deviations for all the groups presented above except the eighth-nerve cases.

It is obvious that for the cases presented here middle-ear pathology does not introduce nonlinear distortion into this conductive system. Frequency distortion is present, of course, because of the changes in mass and stiffness of the system, and is reflected in the changed shape of the sensitivity curve. But, though less sensitive to small amplitudes of vibration, the ossicular system is capable of responding over the same decibel range without overloading. In the light of earlier work on animals, this result is to be expected. Wever, Bray, and Lawrence [8] showed that changes in middle-ear pressure, while affecting sensitivity, had no effect upon the fidelity of response.

If the inner ear becomes involved the threshold of overload is lowered and the range from threshold to overload becomes markedly restricted. There also, obviously, is a relationship to the recruitment phenomenon, since in every case where recruitment occurs there is a lowering of this overload threshold. Detecting the aural harmonics is an actual measure of the ear's distortion characteristics and is certainly a quantitative picture of the distortion that so many patients with inner-ear pathology complain of. With a linear range of only 18 to 24 db. in these two frequencies the ear obviously will not render a very recognizable pattern when fed by a hearing aid or even a loud voice that has a range greater than this.

Interesting speculations can be introduced as to what factors are producing this lowered overload threshold in these ears with inner-ear lesions. Thus in Ménière's disease, which

is represented by the majority of these cases, it is difficult to explain how, without violating mechanical principles, changes in quantity of endolymph can alter the level at which the hair cells overload. Increasing the quantity of endolymph, if it has any effect, would be that of increasing perilymphatic pressure. Existing evidence § indicates that such pressure increases have no effect on the functioning of the sensory cells of the organ of Corti. If such effects could operate, it would seem far more likely that the middle ear, under the conditions of otosclerosis or adhesions, which might have a varied effect at different amplitudes, would have a lowered overload threshold, but such is not the case. It appears more likely that the lowered overload threshold in inner-ear disease is caused by poorly functioning hair cells brought about by some condition which is toxic to them rather than by factors impeding their normal vibration.

It is interesting to go a step further and compare the actual intensity levels at which this overloading occurs. With audiometric zero as a base line rather than the subject's threshold, the mean overload point for normal ears is 49 db. at 1000 cps and 55 db. at 2000 cps.

The intensity level of overload for the middle-ear loss cases is 88 and 99 db. at 1000 and 2000 cps, respectively. These are considerably higher than those figures for the normal and are to be expected in the face of the conductive impairment. The range from threshold to overload is normal but has been shifted to a higher intensity range by the amount of the mean audibility threshold loss.

The intensity levels of overload for the ears with inner-ear involvement are also higher than those for the normal. They are, in fact, almost 20 db. higher: 69 db. at 1000 cps and 73 db. at 2000 cps. This means that the ears with a normal conductive mechanism but with inner-ear pathology and a shorter range from threshold to overload can actually stand more sound before overloading than can the normal ear. The same effect has been observed in records of the cochlear potentials in experimental animals.[8] This can be explained by the "unhealthy hair cell" principle plus the recognized fact that the area of response along the basilar membrane spreads as sound intensity is increased. If a region of hair cells is damaged, or if their presence is spotty, an elevated threshold is produced because a greater spread of response is required to bring into action a sufficient number of hair cells to stimulate an adequate supply of nerve fibers.

Because of the reduced number of hair cells in the region of maximum response their contribution to the entire nerve stimulating effect will be insignificant, so even though there is an overload, the fact may not be recognizable until cells farther out in the area of response add their contribution to the overload effect. This requires more sound, which accounts for the fact that it takes a more intense sound to overload the ear with inner-ear pathology than the normal ear. This by no means indicates that such an ear is safer in a loud sound than a normal ear, because the cells in the region of maximum response, though too few in number to add much to the overload effect, are, nevertheless, being vibrated with sufficient amplitude, possibly to knock them free from the basilar membrane, bringing about a further elevation of the audibility threshold.

SUMMARY

The hearing range from threshold to the beginning of nonlinear distortion was measured in 145 normal ears at 1000 cps and 133 ears at 2000 cps. This was done by using the exploring tone method for detecting what has been called the threshold of aural overload. The extensive individual differences in this range are the same as reported earlier.

By employing the overload threshold measurement technique in abnormal ears it is shown that pathology of the middle ear, while affecting sensitivity, does not alter the very linear characteristics of the conductive mechanism. On the other hand, pathology of the

§ References 6 and 7.

inner ear produces a marked restriction of the hearing range from threshold to overload. The technique of measuring the threshold of aural overload provides a quantitative measure of audible distortion, and the significance of its presence in various inner-ear pathologies is discussed.

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Effects of Testing Methods on Hearing Thresholds

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INTRODUCTION

As reported in Hirsh,[8] "The determination of the [absolute] threshold for a tone of a particular frequency for any one individual is complicated by many problems inherent in psychophysical measurement. . . . The threshold would be different according as the response, the psychophysical technique, and the measurement of the stimulus were of different kinds. Unfortunately, there are not many data that clearly demonstrate these relations. To be sure, the clinical literature is filled with interesting reports of different thresholds measured on the same patients by two operators, by the same operator with different audiometers, etc. But the majority of these involve too many sources of variability at once."

It appears therefore that, while advances in audiometry are both desirable and essential, new techniques should not be generally applied in the clinic until such time as adequate investigations have been conducted to determine the relative merits and limitations of proposed changes in, or additions to, the testing situation. For example, consider the classic psychophysical method of adjustment as applied in the new Békésy-type audiometer.[4] There is little doubt that the method provides a fairly simple and relatively rapid means for the measurement of a hearing threshold. However, there are no data which show the relationship of threshold values obtained by this technique and those obtained by more conventional clinical methods, such as the method of limits. Thus, the feasibility and advisability of using the method of adjustment in clinical applications will depend upon the results obtained in appropriate studies of "comparative audiometry."

PROBLEM

The purpose of the present study was to determine the effects of two audiometric testing methods on absolute thresholds of hearing. Specifically, the study was designed to compare threshold data obtained by the method of limits and the method of adjustment at selected frequencies between 250 and 8000 cps. Three statistical measures were primarily used in the comparison at each frequency: (1) mean sound pressure level (SPL) of absolute thresholds of hearing, (2) variability, among individuals, of absolute thresholds of hearing, and (3) the product-moment correlation between individual thresholds obtained under the two testing conditions. In the analysis of data, a secondary problem was introduced which concerned the evaluation of two techniques of reading the continuous Békésy-type audiogram obtained in the method of adjustment.

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This research was supported by the United States Air Force under Contract AF 33(038)-786, monitored by the Aero-Medical Laboratory, Wright-Patterson Air Force Base. The opinions and conclusions contained in this report are those of the author. They are not to be construed as reflecting the views or endorsement of the Department of the Air Force. Portions of this report were presented at the 49th Meeting of the Acoustical Society of America held at University Park, Pa., on June 29-July 2, 1955.

APPARATUS AND CALIBRATION

The two testing units used in this study were (1) an ADC audiometer, Model 50-B2, and (2) a Békésy-type audiometer, Reger Model. Except for a Brüel and Kjaer beat frequency oscillator, Type 1011, which was substituted for the General Radio beat frequency oscillator, Type 1304-A, the latter audiometer was essentially identical with that described in detail by Reger.[11]

The general procedure in the calibration of the two audiometers involved the measurement of the sound pressure output of each earphone into a National Bureau of Standards Coupler 9-A.[8] With the earphone coupled directly to the cavity * and the coupler microphone placed at the opposite end of the cavity, a given voltage at a particular frequency was applied to the earphone. The sound pressure in the coupler was then determined by measuring the open-circuit output voltage of the coupler microphone.[2]

The general arrangement of the apparatus used in the calibration of the audiometers is presented in the block diagram of Figure 1. For a given audiometer, a frequency setting was selected and the hearing-loss attenuator was set at an appropriate value, usually 60 db. The specific setting of the attenuator was determined by the noise level in the calibrating system and the calibration specifications of the American Standards Association.[1] Switch 1 was closed and the voltage generated in the coupler microphone by the sound pressure in the coupler was read at VTVM-2. Switch 1 was then opened. Switch 2 was closed and the attenuator in the insert circuit was adjusted until the output voltage of the oscillator, as read at VTVM-2, was the same as that previously obtained in response to the output of the audiometer. VTVM-1 was used in monitoring the output of the oscillator to maintain a constant reference level of 1.0 volt.

The sound pressure output of the audiometer into the standard coupler was calculated from the following equation:

$$Lp = 20 \log_{10} \frac{V_1}{V_0} - M_0 + 74$$

where

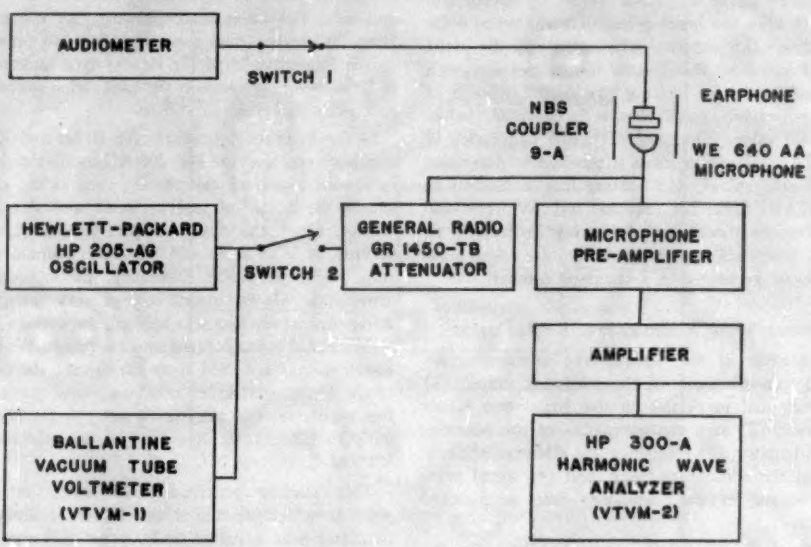

Fig. 1.—Block diagram of apparatus used in the calibration of ADC and Békésy-type audiometers.

Lp = the sound pressure level of the output of the audiometer into the coupler expressed in decibels re 0.0002 dyne/cm.²

V_1 = the open-circuit voltage input to the coupler microphone.

V_0 = the reference voltage (1.0 volt).

M_0 = the pressure response of the coupler microphone in decibels re 1.0 volt per dyne/cm.² as determined by prior calibration.

74 = 1.0 dyne/cm.² in decibels re 0.0002 dyne/cm.²

In solving the preceding equation, the value for the expression $20 \log_{10} \frac{V_1}{V_0}$ was obtained directly

* Such a standard cavity (6 cc.) represents the acoustic impedance that would be presented to the earphone by an average human ear over an appropriate frequency range.

by subtracting the attenuation in the insert circuit from the output voltage of the oscillator read in decibels re 1.0 volt at VTVM-1.

ADC Audiometer, Model 50-E2

It is generally known that experimentally derived minimum audible pressure (MAP) values for the absolute threshold of hearing are consistently lower than the values specified as requirements for acceptable pure-tone audiometers for diagnostic purposes by the Council on Physical Medicine and Rehabilitation of the American Medical Association.[7] Consequently, since the ADC audiometer was to be used for obtaining threshold data on "normal" subjects, the output voltage of the audiometer was adjusted so that the sound pressure of the earphones, as measured in the standard coupler, approximated the MAP values of Sivian and White[12] when the hearing-loss dial was set at 0 db. In addition, an external attenuator (20 db. step) was provided so that, when testing persons with extremely sensitive hearing, the output voltage of the audiometer could be reduced well below threshold value. Thus, the particular calibration of the ADC audiometer made it possible to determine two specific values: (1) hearing loss in decibels re the MAP curve of Sivian and White[12] and (2) absolute thresholds of hearing in decibels re 0.0002 dyne/cm.^{2}, as computed from the sound pressure levels generated in a standard coupler.

Békésy-Type Audiometer, Reger Model

Calibration of the Békésy-type audiometer involved measurements on the following items: (1) frequency-dial markings on the Brüel and Kjaer oscillator, (2) step characteristics of the hearing-loss attenuator, (3) frequency and attenuation markings on the audiogram blank, and (4) sound pressure output of the earphones into a standard coupler.

In the calibration of frequency-dial markings, the output of the Brüel and Kjaer oscillator was compared under load with a laboratory frequency standard † by the observation of Lissajous' figures on a Dumont cathode-ray oscillograph, Type 208-B. Corrective factors for the dial markings were then computed.

In calibrating the attenuator of the audiometer, the apparatus was set up as described in the general calibration procedure. As the setting of the attenuator was discretely changed, the resulting change in output voltage was observed at VTVM-2 (Fig. 1) and measured by attenuation of the insert voltage. It was found that the attenuator did not move in equal steps, nor did it move the same amount when increasing and decreasing in attenuation. However, attenuation was constant as a function of frequency. The data on the calibrated attenuation intervals were used to form two scales: one for increasing attenuation and the other for decreasing attenuation. These scales were transferred to a plastic template, which was later used to read threshold values from the individual Békésy-type audiograms.

Since data on absolute thresholds of hearing were to be obtained in this study, earphone calibration measurements on the Békésy-type audiometer were made in such a manner that threshold readings on the audiogram blanks were in terms of sound pressure re 0.0002 dyne/cm.^{2} for all frequencies, rather than in terms of hearing loss based upon some arbitrarily selected laboratory standard of "normality." This was accomplished by using the "straight" rather than the "curved" drive provided on the Reger model of the Békésy-type audiometer and reversing the steps in the calibration procedure previously described.

In the modified procedure, the Brüel and Kjaer oscillator replaced the HP 205-AG oscillator shown in Figure 1 and was alternately used as the signal source for both the insert circuit and the audiometer. Given the insert reference voltage (1.0 volt as read at VTVM-1) and the desired sound pressure for a particular frequency, the appropriate attenuation of the insert voltage was calculated. After this attenuation was applied, the voltage input to VTVM-2 was observed and recorded. With the insert voltage removed from the circuit, the output of the Brüel and Kjaer oscillator, now serving as the oscillator for the audiometer, was adjusted until an identical voltage reading was obtained at VTVM-2.

This reading provided a calibrated reference point to which the Brüel and Kjaer (audiometer) oscillator was adjusted daily, prior to testing the first subject and after permitting the equipment to "warm up" for at least 30 minutes. Calibration measurements of the sound pressure levels generated by the Permoflux (PDR-8) earphones of the Békésy-type audiometer into the standard coupler were obtained with the attenuator of the audiometer set at 50 db. SPL on the audiogram blank.‡

By replicating the procedures described for the calibration of frequency and attenuation, a master audiogram blank was obtained which provided calibrated reference lines for the measurement of sound pressure and frequency. The blank was then reproduced in sufficient quantity for the entire experiment.

† The laboratory frequency standard was a unit constructed and calibrated by the Department of Physics, The Pennsylvania State University.

‡ For 1000 cps this value approximates a hearing loss of 40 db., based on the curve of Sivian and White.[12]

PROCEDURE

Subjects

The subjects in this study were members of an introductory course in psychology at The Pennsylvania State University. Both white men and women between the ages of 17 and 25 years, inclusive, participated in the study. No attempt was made to select subjects; neither those having well-defined hearing losses nor those having a history of excessive exposure to noise were excluded. In all, 105 subjects were tested, with 75 males and 30 females. Prior to the first audiometric test, each subject was given a complete otological examination.§ In the examination a National-Fontar nasopharyngoscope, No. 7 F., was used, and the results of the examination were recorded on an appropriate form. Any foreign body, including excessive cerumen, found in the external auditory canal was appropriately removed before the subject was given the first audiometric test. All subjects were individually tested in an anechoic chamber which was provided with a two-way voice communication system.[5]

Test on ADC Audiometer

As soon as possible after the otological examination was performed, usually within 24 to 48 hours, each subject was given a test on the ADC audiometer to determine his monaural threshold of hearing for air-conducted sound for both right and left ears. Within the group, the order of testing right and left ears was varied more or less at random.

Prior to each test, the following instructions were read to the subject after he had seated himself comfortably in a chair in the testing room:

"I am going to run a test to find out how 'soft' a tone you can hear. The tones you will hear will be different in pitch; some will sound like very high whistles, others will sound like fog-horns or low notes on a piano. Each tone will be turned on and off several times. Sometimes the sound will get weaker and weaker until you can hear it no longer; at other times, it will get louder and louder until you do hear it. Whenever you hear a sound come on, press this button [tester shows key to subject] with your thumb and hold it down until the sound stops. This sign [tester points to sign] will remind you of these instructions, if you should forget what you are to do. After I have placed the earphones on your head, do not touch them in any way. Are there any questions?"

The tester then removed any eyeglasses worn by the subject and, for female subjects, pushed the hair away from the ears. The earphones were next positioned and centered over the ears with enough tension in the headband spring to hold the earphones firmly in place throughout the test.

The specific testing technique used to obtain threshold data on the ADC audiometer represented a modification of the classical method of limits. The first step was to present a 1024 cps tone in the right (or left) ear at a setting of 30 db. on the hearing-loss dial. If the subject did not report hearing this, the intensity was increased in steps of

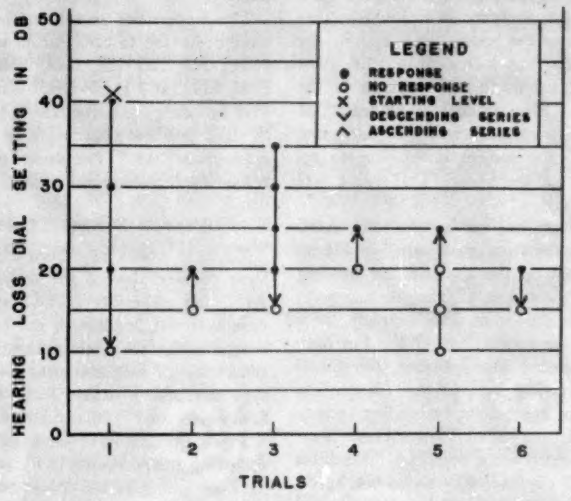

Fig. 2.—Threshold testing procedure used with ADC audiometer.

§ The otological examinations were performed by two physicians of the Health Service, The Pennsylvania State University.

10 db. until the subject gave a positive response on a signal light. When the tester was certain that the subject heard the test tone, as indicated by repeated responses to the randomly interrupted tonal signal, he decreased the intensity in 10 db. steps until the subject's first failure to respond to the onset of the tone occurred. (This value was recorded for Trial 1.) The tester then increased the signal intensity in 5 db. steps until the subject made his first response. (This value was recorded for Trial 2.) The 1024 cps tone was again presented, but at a level only 15 db. above the recorded value for Trial 2. The signal intensity was decreased in 5 db. steps until the subject again failed to respond once to the onset of the tone. (This value was recorded for Trial 3.) The tester then increased the signal intensity in 5 db. steps until the subject made one response. (This value was recorded for Trial 4.) Starting at an intensity setting of 15 db. below the value for Trial 4, the intensity of the signal was increased in 5 db. steps until the subject again made one response to the onset of the tone. (This value was recorded for Trial 5.) The signal intensity was then decreased in 5 db. steps until the subject failed once to respond to the onset of the tone. (This value was recorded for Trial 6.)

The procedure used in testing with the ADC audiometer is diagrammatically presented in Figure 2, with trials plotted on the abscissa and hearing loss in decibels on the ordinate. The starting point for Trial 1 (X) is shown, in this example, as a setting on the hearing-loss dial of 40 db. For each trial, the arrowhead indicates whether the stimuli presented are decreasing in intensity (descending series) or increasing in intensity (ascending series). Also for each trial, the stimulus intensities (hearing-loss settings) which were heard by the subject are coded as filled circles (●); those not heard, by unfilled circles (○). Notice that in all trials except the first the hearing-loss dial is moved in 5 db. steps. Furthermore, there are three descending and three ascending series, with Trials 3 and 5 starting, respectively, 15 db. above and below Trials 2 and 4. In each trial, only one response is made or not made to each stimulus presentation.

To compute the stimulus threshold, the recorded values for Trials 2 through 5 are summed and averaged.‖ For the example given in Figure 2, these values are, in trial order, 20, 15, 25, 25, and 15, which yield a sum of 100 and a mean threshold (hearing-loss) value of 20 db. The estimate of the threshold of hearing obtained by this method of analysis is within 0.5 db. of the threshold computed by the traditional technique of the method of limits.[18] For each frequency tested, the hearing-loss value was converted to an absolute sound-pressure value by adding an appropriate correction factor as determined by the prior calibration of the earphones in the standard coupler.

Although the testing technique described for the ADC audiometer is not that specifically used by audiometrists, it does follow the usually accepted procedure of starting well above threshold and crossing threshold a number of times in each direction for a given frequency.[8] Furthermore, the procedure, when precisely followed, tends to minimize the variability in threshold results for a given subject tested by different examiners. It also provides a specific criterion of response or nonresponse for a given trial and, for each frequency, produces a threshold value based upon the same number of trials.

The frequencies tested on the ADC audiometer were presented to each ear in the following order: 1024,¶ 512, 256, 128, 1024, 1448, 2048, 2896, 4096, 5792, 8192, and 11,584 cps. When the tests on the first ear were completed the subject was instructed, "I shall now test your [right or left, whichever was appropriate] ear." The same testing procedure and frequency sequence was used in all threshold tests.

Test on Békésy-Type Audiometer

While it would have been extremely desirable to have counterbalanced the order of testing on the ADC and Békésy-type audiometers, such a procedure was not possible in the present study due to time considerations and calibration difficulties. Consequently, all subjects were tested on the Békésy-type audiometer after their tests on the ADC audiometer had been completed.

Prior to each test, each subject was read the following instructions:

"I am going to run a test to find out how 'soft' a tone you can hear. I will first test your right ear, then your left. The tone you will hear will start out very 'low'; that is, it will sound something like a low note on the piano. The tone will then get gradually higher and higher in pitch. Your task is to adjust the loudness of this tone so that it is always just barely audible. If you want the tone to get 'softer' or 'fade away,' just press this switch # and hold it down. This will cause the tone to get so 'soft' that you will no longer be able to hear it. So, in order to get the tone louder again, release the switch. The tone will then gradually 'build up.' You must listen very carefully, pressing

‖ The threshold estimate of Trial 1 is not used in this computation, since the hearing-loss dial was moved in 10 db. steps. This trial serves only to locate quickly the general region of hearing.

¶ The initial threshold test at 1024 cps was considered a practice trial, and the midtest 1024 cps threshold value was taken as the "correct" value in all subsequent computations.

The subject was provided with a General Electric mercury switch, Type 2-52KR1, mounted so as to operate when tilted 15 degrees.

and releasing the switch in such a way that the tone is always just barely audible. Remember, to make the tone get 'softer,' press the switch; to make the tone get louder, release the switch. This sign [tester points to sign] will remind you of these directions. Are there any questions? [Pause.] After I have placed the earphones on your head, do not touch them in any way. If you should need to talk to me for any reason, just speak up and I will hear you over this microphone."

After the necessary precautions about hair and eyeglasses had been taken, the tester positioned the earphones on the subject's head and returned to the control room. The subject was then instructed, "The test will now begin. I will test your right ear first."* The auditory signal was always introduced at a frequency of 200 cps, 45 db. SPL, and terminated just beyond 8000 cps.

After the test for the right ear was completed, the subject was instructed, "That is all for that test, but do not touch your earphones. I will now open the chamber doors and you may relax for five minutes." At the end of that time, during which the tester made appropriate adjustments on the audiometer, the subject was instructed, "I will now test your hearing on your left ear. Follow the same instructions as before. Press the switch to make the tone 'softer,' release the switch to make the tone louder. The test will now begin." The test for the left ear followed exactly the same procedure as that for the right.

All tests on the Békésy-type audiometer were conducted using a testing time of 10 minutes per ear and an attenuation rate of 0.5 db. per second. These values were based on the results of a previous study which indicated that this combination of operating conditions produces minimal threshold variability and maximal test-retest reliability.[6]

RESULTS

In attempting to extract threshold data from the audiograms obtained on the Békésy-type audiometer, an important question was raised. Specifically, where on the audiogram trace is the threshold curve? Békésy [4] mentions that "the tone oscillates around the threshold of the subject the whole time," but does not indicate exactly how the audiogram should be read. Reger [11] and, later, Corso [6] both reported making threshold measurements by using "the curve connecting the midpoints between the peaks and valleys of the ink-recorded audiogram." Consequently, in the first attempt at reading the Békésy-type audiograms in the present study, threshold readings were determined by the intersection of this "midpoint" curve and specific frequency lines.

An inspection of the data obtained in this manner suggested, however, that the "midpoint" curve was probably not a valid estimate of the "true" threshold curve, since the obtained threshold sound pressure levels appeared to be somewhat greater than those obtained on the ADC audiometer for the same subjects. Therefore, an alternative method of reading the Békésy-type audiogram was devised.

In this method, the threshold curve for a given subject was designated as the curve connecting the "peaks" of the ink-recorded audiogram, where the "peaks" are defined as the points of transition at minimal values of sound pressure. Threshold readings were then determined by the intersection of the "peak" curve and specific frequency lines.

Figure 3 presents a graphic illustration of the two types of curves used in reading the Békésy-type audiogram. The audiogram shown contains the right ear data of a typical subject and covers the frequency range between 250 and 8000 cps. In Part *A* of Figure 3, the "midpoint" curve is represented by a dashed line connecting "the midpoints between the peaks and valleys of the ink-recorded audiogram." In Part *B* of Figure 3, the "peak" curve is represented by a dashed line connecting "the points of transition at minimal values of sound pressure." In both parts of Figure 3, threshold is taken as the point of intersection of the appropriate curve with a specific frequency line. For example, for 2000 cps, the threshold value is 23.0 db. as determined by the "midpoint" curve and 16.0 db. as determined by the "peak" curve. Obviously, for a given audiogram, the "peak" reading will always be smaller than the "midpoint" reading.

* In the latter part of the study, when the testers had gained sufficient experience in manipulating the Békésy-type audiometer, the order of testing ears was alternated between right and left. However, most subjects in this study were tested first on the right ear.

Table 1 presents a summary of the analysis performed to determine the effects of reading the Békésy-type audiogram by the two techniques described: "midpoint" curve and "peak" curve. For 105 subjects tested monaurally on the Reger model of the Békésy-type audiometer, means and standard deviations were computed for threshold sound pressure levels at nine frequencies for each ear. Tests of significance were then performed to determine whether the obtained differences in both means and standard deviations at each frequency were statistically significant, i. e., not due to chance factors. The results of these tests showed that for either ear at no frequency is there a significant difference in variability (F-ratio), but each difference in means (t-ratio) is significant beyond the 1% level, i. e., could only have occurred by chance less than once in a hundred times.

The two sets of threshold data ("midpoint" and "peak") obtained on the Békésy-type audiometer were then compared with the results of the tests on the ADC audiometer. This comparison was made for two purposes: (1) to determine the effects of the testing method (method of adjustment and method of limits, respectively) on absolute thresholds of hearing and (2) to determine whether the measurements based upon the "midpoint" curve or "peak" curve were more valid estimates of absolute thresholds of hearing, using the ADC values as criterion scores.

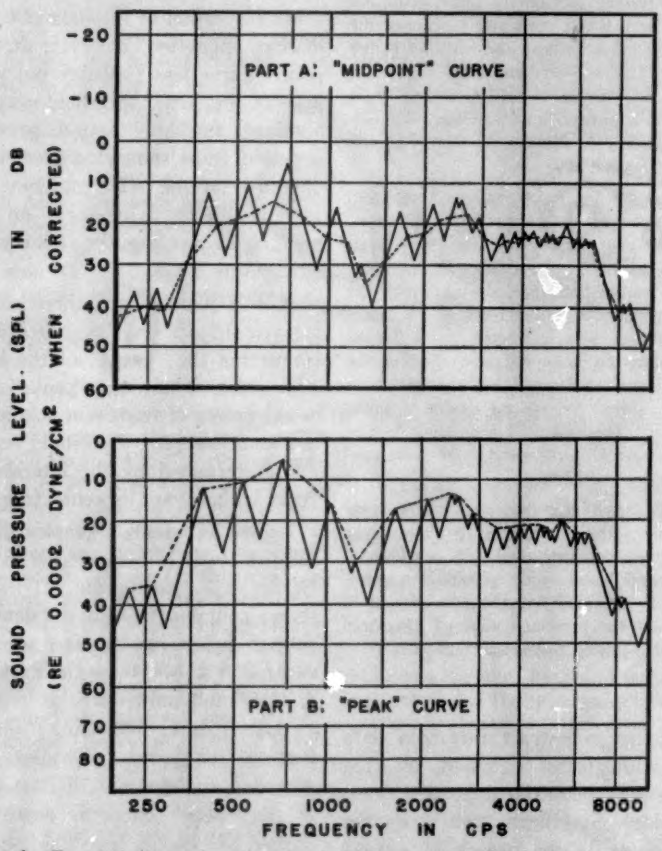

Fig. 3.—Two techniques of reading threshold data from the Békésy-type audiogram.

TABLE 1.—*Comparison of Threshold Intensities Obtained by "Midpoint" and "Peak" Techniques on the Békésy-Type Audiogram*

Measurement Technique	Frequency in Cps	Mean Threshold SPL in Db. re 0.0002 Dyne/Cm.²	*t*-Ratio*	Standard Deviation in Db.	*F*-Ratio
		Left Ear (N-105)			
Midpoint	250	32.8	7.32	8.51	1.05
Peak		30.1		8.30	
Midpoint	500	18.2	12.26	7.74	1.25
Peak		9.1		8.66	
Midpoint	1000	7.0	22.22	9.54	1.25
Peak		3.8		10.67	
Midpoint	1500	7.9	9.25	11.64	1.14
Peak		4.7		12.45	
Midpoint	2000	10.4	27.80	10.39	1.30
Peak		6.5		11.72	
Midpoint	3000	15.2	8.18	11.28	1.10
Peak		12.5		10.78	
Midpoint	4000	13.7	9.36	12.02	1.09
Peak		9.9		13.47	
Midpoint	6000	31.4	7.14	14.50	1.02
Peak		26.4		14.38	
Midpoint	8000	29.6	8.88	14.94	1.04
Peak		17.4		15.24	
		Right Ear (N-105)			
Midpoint	250	40.9	6.00	10.39	1.06
Peak		37.9		10.10	
Midpoint	500	17.8	12.26	8.01	1.09
Peak		14.0		7.67	
Midpoint	1000	8.4	38.00	6.84	1.04
Peak		4.6		6.70	
Midpoint	1500	8.5	8.35	7.83	1.03
Peak		4.5		7.93	
Midpoint	2000	10.1	12.31	8.84	1.07
Peak		6.9		9.13	
Midpoint	3000	14.6	10.18	9.81	1.10
Peak		11.1		10.31	
Midpoint	4000	14.4	6.78	10.58	1.11
Peak		10.6		11.15	
Midpoint	6000	31.6	5.69	13.21	1.04
Peak		27.9		13.15	
Midpoint	8000	21.3	8.08	13.96	1.07
Peak		17.1		14.50	

* All *t*-ratios in the column are statistically significant at the 1% level.

Table 2 presents a summary of the comparison of the absolute thresholds obtained for the 105 subjects on the ADC audiometer and on the Békésy-type audiometer with "midpoint" readings. The upper portion of Table 2 contains the data for the left ear; the lower portion, for the right ear. In both portions of the table, the mean and standard deviation of the 105 threshold values are given for each of nine frequencies.† Also shown for each frequency are the results of the test of significance of the differences in means (*t*-ratio), the results of the test of significance of the differences in variability (*F*-ratio), and the Pearson product-moment correlation coefficient (*r*).[10]

Inspection of Table 2 shows that, for the left ear, six of the nine differences in means (*t*-ratios) are statistically significant at or beyond the 5% level; for the right ear, eight of the nine differences are significant at or beyond the 1% level. In either ear, where statistically significant differences in means were obtained, the mean absolute threshold values for the ADC audiometer are consistently smaller than the values for the Békésy-type audiometer.

In variability, the left ear shows five frequencies (250 to 2000 cps, inclusive) at which the difference (*F*-ratio) is statistically

† The frequencies indicated for both ADC and Békésy-type audiometers are the calibrated, not nominal, values. While slight discrepancies exist between the frequencies of the two audiometers, these discrepancies are less than 2% and should not appreciably affect the results or conclusions of this study.

TABLE 2.—*Comparison of Threshold Intensities Obtained on ADC and Békésy-Type Audiometers ("Midpoint" Measurements)*

Type of Audiometer	Frequency in Cps	Mean Threshold in Db. SPL re 0.0002 Dyne/Cm.²	*t*-Ratio	Standard Deviation in Db.	*F*-Ratio	Correlation Coefficient (*r*)
			Left Ear (N-105)			
Békésy	250	32.8	12.30*	8.51	2.40*	0.31
ADC	252	22.4		5.49		
Békésy	500	18.2	2.13†	7.74	1.65*	0.28
ADC	504	11.5		6.02		
Békésy	1000	7.0	0.61	9.54	2.82*	0.41
ADC	1000	6.5		5.68		
Békésy	1500	7.9	1.53	11.64	2.62*	0.4
ADC	1410	6.3		7.19		
Békésy	2000	10.4	3.06*	10.39	2.03*	0.39
ADC	1970	7.4		7.29		
Békésy	3000	15.2	11.01*	11.28	1.09	0.67
ADC	2780	5.1		11.78		
Békésy	4000	18.7	0.20	12.92	1.37	0.64
ADC	3940	18.9		15.11		
Békésy	6000	31.4	16.10*	14.50	1.12	0.74
ADC	5600	15.3		13.68		
Békésy	8000	20.6	3.56*	14.94	1.23	0.72
ADC	7800	16.4		16.56		
			Right Ear (N-105)			
Békésy	250	40.9	18.15*	10.30	3.06*	0.16
ADC	252	26.6		5.92		
Békésy	500	17.8	5.71*	8.01	1.60*	0.19
ADC	504	12.6		6.23		
Békésy	1000	8.4	2.85*	6.84	1.51†	0.27
ADC	1000	6.3		5.59		
Békésy	1500	8.5	3.36*	7.83	1.36	0.45
ADC	1410	6.0		6.72		
Békésy	2000	10.1	5.71*	8.84	1.52†	0.57
ADC	1970	5.8		7.16		
Békésy	3000	14.6	6.78*	9.81	1.05	0.51
ADC	2780	7.9		10.05		
Békésy	4000	14.4	1.02	10.58	1.33	0.39
ADC	3940	15.7		12.21		
Békésy	6000	31.6	18.35*	13.21	1.08	0.67
ADC	5600	15.7		13.04		
Békésy	8000	21.3	5.30*	13.96	1.27	0.50
ADC	7800	13.5		15.66		

* *F*-ratio or *t*-ratio significant at 1% level.
† *F*-ratio or *t*-ratio significant at 5% level.

significant at or beyond the 5% level; for the right ear, four of the same five frequencies show differences which are statistically significant at or beyond the 5% level. In each instance, the standard deviation of the 105 threshold values obtained on the ADC audiometer is smaller than the value obtained on the Békésy-type audiometer.

With respect to the degree of correlation between threshold values obtained on the ADC and Békésy-type audiometers, the data of Table 2 indicate that all correlation coefficients for the left ear, and all but two (0.16 and 0.19) for the right ear, are statistically significant at the 1% level. The obtained correlation coefficients ranged from 0.31 to 0.74 for the left ear and from 0.16 to 0.57 for the right ear.

Table 3 presents a summary of the comparison of the absolute thresholds obtained for the 105 subjects on the ADC audiometer and on the Békésy-type audiometer with "peak" readings. The columnar entries of Table 3 are exactly the same as those for Table 2.

Inspection of Table 3 shows that, for the left ear, six of the nine differences in means (*t*-ratios) are statistically significant at or beyond the 5% level; for the right ear, seven of the nine differences are significant at or beyond the 5% level. In either ear, where statistically significant differences in means were obtained, there is no consistent trend for the absolute threshold values of the ADC audiometer to be either larger or smaller

TABLE 3.—*Comparison of Threshold Intensities Obtained on ADC and Békésy-Type Audiometers ("Peak" Measurements)*

Type of Audiometer	Frequency in Cps	Mean Threshold in Db. SPL re 0.0002 Dyne/Cm.²	*t*-Ratio	Standard Deviation in Db.	*F*-Ratio	Correlation Coefficient (*r*)
			Left Ear (N-105)			
Békésy	250	30.1	9.35*	8.30	2.29*	0.32
ADC	252	22.4		5.49		
Békésy	500	9.1	2.85*	8.66	2.07*	0.36
ADC	504	11.5		6.02		
Békésy	1000	3.8	2.75*	10.67	3.58*	0.37
ADC	1000	6.5		5.98		
Békésy	1500	4.7	1.42	12.45	3.00*	0.42
ADC	1410	6.8		7.19		
Békésy	2000	6.5	0.02	11.72	2.56*	0.42
ADC	1970	7.4		7.29		
Békésy	3000	12.5	8.36*	10.78	1.19	0.03
ADC	2780	5.1		11.78		
Békésy	4000	9.9	3.00*	12.47	1.36	0.59
ADC	3940	13.8		15.11		
Békésy	6000	20.4	8.87*	14.28	1.10	0.59
ADC	5600	15.8		15.68		
Békésy	8000	17.4	0.81	15.24	1.18	0.69
ADC	7800	16.4		16.56		
			Right Ear (N-105)			
Békésy	250	37.9	10.75*	10.06	2.95*	0.11
ADC	252	26.6		5.92		
Békésy	500	14.0	1.52	7.67	1.47†	0.23
ADC	504	12.7		6.88		
Békésy	1000	4.6	2.34†	6.70	1.45†	0.30
ADC	1000	6.8		5.56		
Békésy	1500	4.3	2.18†	7.98	1.39†	0.52
ADC	1410	6.0		6.72		
Békésy	2000	6.9	1.43	9.18	1.63*	0.56
ADC	1970	5.8		7.16		
Békésy	3000	11.1	3.06*	10.81	1.06	0.48
ADC	2780	8.0		10.06		
Békésy	4000	10.6	4.48*	11.15	1.20	0.50
ADC	3940	15.7		12.21		
Békésy	6000	27.9	8.66*	13.15	1.02	0.40
ADC	5600	15.7		13.04		
Békésy	8000	17.1	2.34†	14.50	1.17	0.45
ADC	7800	13.5		15.06		

* *F*-ratio or *t*-ratio significant at 1% level.
† *F*-ratio or *t*-ratio significant at 5% level.

than the values for the Békésy-type audiometer.

In variability, both the left and right ears show statistically significant differences, at or beyond the 5% level, for the five lowest frequencies tested (250 to 2000 cps, inclusive). In each instance, the standard deviation of the 105 threshold values obtained on the ADC audiometer is smaller than the value obtained on the Békésy-type audiometer.

In terms of the correlation between the two sets of threshold values at each frequency, the data of Table 3 indicate that all correlation coefficients for the left ear, and all but two (0.11 and 0.23) for the right ear, are statistically significant at the 1% level. The obtained correlation coefficients ranged from 0.32 to 0.69 for the left ear and from 0.11 to 0.56 for the right ear.

The final analysis of data was performed to determine whether the differences between the correlations of the "peak" and "midpoint" threshold values with the ADC threshold values were statistically significant. The results of this analysis are presented in Table 4, together with specific correlation coefficients obtained at each frequency for each ear. The *t*-tests of significance [9] show that for the left ear there is a statistically reliable difference, at or beyond the 5% level, in the correlation coefficients at both 4000 and 6000 cps. In each of these two instances, the correlation coefficient based upon the "midpoint" readings was greater than that based upon "peak" readings. For the right

TABLE 4.—*Comparison of Correlation Coefficients Obtained Between Threshold Intensities on ADC and Békésy-Type Audiometers*

Frequency in Cps	Correlation Coefficient (r_{12}) for Békésy "Midpoint" and ADC Thresholds	Correlation Coefficient (r_{13}) for Békésy "Peak" and ADC Thresholds	Correlation Coefficient (r_{23}) for Békésy "Midpoint" and "Peak" Thresholds	t-Ratio for r_{12}-r_{13}
		Left Ear (N-105)		
250	0.31	0.32	0.99	−0.241
500	0.33	0.36	0.92	−0.810
1000	0.41	0.37	0.90	0.999
1500	0.44	0.42	0.96	0.801
2000	0.39	0.42	0.94	−0.960
3000	0.67	0.68	0.95	0.434
4000	0.64	0.59	0.95	2.064*
6000	0.74	0.59	0.88	4.750†
8000	0.72	0.69	0.97	1.774
		Right Ear (N-106)		
250	0.16	0.11	0.87	0.900
500	0.19	0.23	0.92	−1.044
1000	0.27	0.30	0.99	−2.331*
1500	0.45	0.52	0.81	−1.900
2000	0.57	0.56	0.96	0.436
3000	0.51	0.45	0.94	1.032
4000	0.39	0.50	0.86	−2.440*
6000	0.57	0.49	0.87	4.269†
8000	0.50	0.45	0.93	1.244

* t-ratio significant at 5% level.
† t-ratio significant at 1% level.

ear, statistically significant differences, at or beyond the 5% level, were found at 500 cps and, again, at 4000 and 6000 cps. However, only the "midpoint" correlation coefficient at 6000 cps was larger than those based upon "peak" readings of the Békésy-type audiograms.

COMMENT

The results of Table 1 comparing the absolute thresholds of hearing based upon "midpoint" and "peak" readings of the Békésy-type audiograms clearly indicate that the technique of reading the audiogram will significantly displace the mean threshold of hearing curve without affecting the group variability at any given frequency. However, since the magnitude of the discrepancy in mean sound pressure level for a given frequency is never greater than 5 db., it does not appear that the difference introduced by the two techniques of reading the audiogram has any practical significance. Thus, it would appear that the professional examiner might select either method for clinical use, provided the instructions to the subject are not appreciably altered from those used in the present study. These instructions emphasize that the loudness of the tone is to be adjusted so that "it is always just barely audible." It is believed that this terminology is more understandable to the unsophisticated listener than the concept of "crossing threshold" and should be retained in future tests of this type.

It also follows from the specific wording of these instructions that the listener would not be expected to provide an audiogram trace which "crosses threshold." This view appears to be supported by the data of Table 2, which show that, in general, the threshold intensities for the ADC audiometer are consistently lower than those for the "midpoint" readings on the Békésy-type audiometer. If the listener followed his instructions carefully, the tone would always be "just barely audible" and his audiogram trace would "go below threshold" only by chance. Consequently, the best estimate of the "traditional" threshold in this case would be that based upon the "peak" values of the audiogram. Furthermore, if the ADC and Békésy "peak" values are both measures of "true" threshold, no systematic trend in the differences in means should occur as a function of fre-

quency. Inspection of the data of Table 3 shows that this is a tenable hypothesis inasmuch as no consistent trend can be detected in the relative sound pressure magnitudes of absolute thresholds under the two testing conditions.

One observation in the comparison of the specific methods of this study appears fairly well established: For the sample of subjects tested, the method of limits produces significantly less variability among absolute threshold intensities than the method of adjustment, at least for those frequencies below 2000 cps. This is evident in the data of Tables 2 and 3. Since the variability at a given frequency represents differences in thresholds among individuals, this might be interpreted as an indication that the test on the ADC audiometer does not discriminate as well as the test on the Békésy-type audiometer, at least at the lower frequencies.

Inspection of the data in Tables 2 and 3 reveals a trend in correlation which may be associated with this effect. As frequency increases, the correlation coefficients between threshold intensities on the ADC audiometer and threshold intensities for both "midpoint" and "peak" readings on the Békésy-type audiometer tend to increase, although not in an entirely consistent manner. It is possible that the explanation of this trend may be related to the obtained variability among individuals. At the lower frequencies, where the ADC test may not differentiate among individual thresholds, the correlation coefficients are low. However, as the frequency increases, the variability among individuals on the ADC test becomes larger and may, consequently, produce higher correlation coefficients. This hypothesis appears tenable, since the data in Tables 2 and 3 show that the highest correlation coefficients occur, in general, at those frequencies at which variability among individuals is greatest.‡

The largest discrepancies in mean threshold intensities for the two psychophysical methods appear in Tables 2 and 3 at frequencies of 250 and 6000 cps. For 250 cps, the discrepancy is attributed, in part, to the specific starting level used in the method of adjustment; this level was 40 db. SPL at a frequency of 200 cps. Since the attenuation rate used in testing was 0.5 db/sec. and the testing time was approximately 2 min/octave, threshold intensity for a given subject might not have been reached by the time the frequency dial had moved to 250 cps, even though attenuation was increasing constantly from the start of the test. If the subject initially decreased the attenuation, either by error or to hear the signal better, the intensity of the signal at 250 cps would probably be well above threshold. The unusually large threshold values at 250 cps for the right ear are most likely due to lack of familiarity with the testing procedure. It should be recalled that most subjects were tested first on the right ear when the Békésy-type audiometer was used.

The discrepancies in mean threshold intensities at 6000 cps may, perhaps, be explained in terms of acoustic resonance produced in the standard coupler by the earphones of the

‡ To provide a preliminary check on the foregoing hypothesis, nine subjects were tested and retested on the right ear by using the method of adjustment. For 500 cps, a correlation coefficient was computed between the "peak" readings of the test audiograms and the "midpoint" readings of the retest audiograms. If the hypothesis is assumed to be "true," then the obtained correlation coefficient should be greater than that for either the "midpoint *vs.* ADC" value (0.19) or "peak *vs.* ADC" value (0.23). The obtained correlation coefficient was 0.84 and is statistically significant at the 1% level. While this evidence is not conclusive, it does support the notion that, at the lower frequencies, the Békésy-type audiometer does discriminate better among individual thresholds than does the ADC audiometer.

The reader is reminded that, although statistically significant (i. e., greater than zero) positive correlations were obtained at nearly all frequencies, these correlation coefficients are too low to be of real value in predicting, for a given frequency, a subject's ADC threshold from his "midpoint" or "peak" threshold on the Békésy-type audiometer. Before the error of estimate in predicting ADC thresholds from Békésy thresholds, or vice versa, is reduced by one-half, the correlation coefficient (r) must be as high as 0.866.[10]

Békésy-type audiometer during the sound pressure calibration measurements. At frequencies up to approximately 5000 or 6000 cps, the response of the coupler agrees closely with the response of the human ear. At frequencies higher than this, the ear is found to have sharp resonances which are not duplicated in the coupler. Thus, the mean sound pressure values of absolute threshold given in Tables 2 and 3, while taking into account coupler calibration measurements, may not accurately indicate the actual sound pressure at the eardrum.[8]

The effects of practice on the absolute thresholds of hearing obtained on the Békésy-type audiometer can be seen in Tables 2 and 3. As mentioned, most subjects were tested first on the right ear. Inspection of the data shows that there is a noticeable decrease in the mean sound pressure level for both "midpoint" and "peak" thresholds for the second ear tested (left). Also, the data of Table 4 show that there is an increase in the reliability of the threshold measurements from right to left ears, as indicated by somewhat larger correlation coefficients for the left ear. It would appear advisable, therefore, that in future tests on the Békésy-type audiometer, the subject be permitted a few minutes of practice in which to listen to, and adjust, the tonal signal.

SUMMARY AND CONCLUSIONS

Absolute thresholds of hearing from 250 to 8000 cps were obtained on 105 male and female subjects between the ages of 17 and 25 years, inclusive, by the method of limits and the method of adjustment. The specific instruments used in each of these two methods were, respectively, an ADC audiometer, Model 50-E2, calibrated on the Sivian and White [12] curve, and the Reger model of the Békésy-type audiometer, calibrated in terms of sound pressure level re 0.0002 dyne/cm.^{2} All subjects were monaurally tested in an anechoic chamber on both right and left ears and, prior to the first audiometric test, were given a thorough otological examination by trained physicians.

In the treatment of data, two techniques of reading the Békésy-type audiogram were investigated: (1) the "midpoint" technique, in which the absolute threshold curve was taken as the curve connecting "the midpoints between the peaks and valleys" of the audiogram, and (2) the "peak" technique, in which the absolute threshold was taken as the curve connecting the "points of transition at minimal values of sound pressure" on the audiogram. The results of this analysis showed that the threshold of hearing curve is significantly lowered by the "peak" technique but the variability of absolute thresholds among individuals is not affected at the frequencies tested.

The two sets of absolute threshold data obtained on the Békésy-type audiometer ("midpoint" and "peak") were then compared with the results of the tests on the ADC audiometer. Three statistical measures were used in the comparison at each of nine frequencies: (1) mean absolute thresholds in decibels SPL, (2) variability of absolute thresholds among individuals, and (3) product-moment correlations between individual thresholds obtained under the two psychophysical procedures. For each of these measures, appropriate tests of statistical significance were used.

The conclusions of this study indicate the following: 1. In general, the method of limits produces mean absolute threshold values that are significantly smaller than those obtained by "midpoint" audiogram readings in the method of adjustment. 2. From 250 to 2000 cps, inclusive, the method of limits produces significantly less variability in absolute thresholds among individuals than either the "midpoint" or "peak" audiogram readings in the method of adjustment. 3. For the frequencies tested, there is no consistent trend for the mean absolute thresholds obtained by the method of limits to be either significantly larger or significantly smaller than the values obtained by "peak" audiogram readings in the method of adjustment. 4. In general, there is a statistically significant, but rather low, positive correlation between individual thresholds obtained at a given frequency by

the method of limits and those obtained by the method of adjustment, using either "midpoint" or "peak" readings on the Békésy-type audiograms.

Pennsylvania State University.

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Case Reports

A LARGE TRACHEAL FOREIGN BODY

CAPT. JAMES L. SHEEHY (MC), U. S. Army

FROM A review of the standard texts of otolaryngology and bronchoesophagology and conversations with various otolaryngologists, tracheobronchial foreign bodies of the size to be reported have been seen rarely. Even more unusual is the fact that the patient survived this episode.

REPORT OF CASE

A 39-year-old nurse was brought into the emergency room after having aspirated a piece of meat 10 minutes earlier. She was immediately taken to the Ear, Nose and Throat Surgery Section.

Five minutes after arrival she appeared pale and apprehensive but was cooperative, and held the upper part of her body inclined forward at a 45 degree angle. Her voice was clear but weak, and there was no stridor or wheezing. She did not wear a dental prosthesis.

Examination of the larynx and hypopharynx by mirror was negative, and there was no pooling of saliva. Her pulse was approximately 100, her blood pressure normal, and since the patient had survived 15 to 20 minutes there seemed to be need for extreme haste. Oxygen by face mask, held by the patient, was started, and intermittently the pharynx and hypopharynx were sprayed with a 5% cocaine solution. Preparations were made for laryngoscopy, bronchoscopy, or tracheotomy and an anesthetist called to assist if necessary.

The patient stated that she had been eating a piece of steak and that while doing so had scolded one of her children sternly. At that point she lost control of the meat and believed she had aspirated it. Her husband stated that immediately there had been an episode of coughing and wheezing but both had ceased shortly before she arrived at the hospital. The patient stated it felt as though the meat was in her windpipe and pointed to the area of the cricoid cartilage.

It was believed that any piece of meat small enough to have passed the larynx certainly would have dropped into one or the other main-stem bronchus, but examination of the chest was negative, and a bronchoscopy was decided upon.

The patient was wheeled into the operating room and given 100 mg. of secobarbital (Seconal) sodium intravenously. During this entire time she maintained the above-described position, but as soon as the secobarbital had taken effect she was turned onto her back and a Jackson laryngoscope was inserted. At this point a small piece of tissue could be seen at the interarytenoid area, just below the level of the true cords. This was grasped with a laryngeal forceps, and a piece of meat measuring 5 by 4 by 3 cm. was delivered (Figure).

The patient's respiratory difficulty immediately subsided, and after a brief rest on the operating-room table she was sent home. The entire elapsed time between lodgment and dislodgment of the foreign body was slightly over 30 minutes. There was no hoarseness the following day.

Fixed specimen.

Accepted for publication June 3, 1955.

Otolaryngology Service, Department of Surgery, Letterman Army Hospital, San Francisco.

NEURILEMMOMA OF THE PHARYNX

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and
JOSE PICÓ, M.D., San Juan, Puerto Rico

TUMORS of the pharynx are more frequently malignant than benign. Neurilemmomas are relatively uncommon. Not a single case of primary pharyngeal neurilemmoma was encountered among 63 benign tumors of the pharynx and tonsil collected by New and Childrey [1] at the Mayo Clinic over a 13-year period. None has been found by one of us (R. A. M.-R.) among 70,000 surgical specimens examined at the Department of Pathology of the University of Puerto Rico Medical School, and the tumor in the present case is the first of its kind among 11,000 specimens, mostly tumors, removed at the I. González Martinez Oncologic Hospital.

Accepted for publication Aug. 15, 1955.

From the Department of Pathology and Otorhinolaryngology of the University of Puerto Rico Medical School and the Isaac González Martínez Oncologic Hospital.

The first relatively large group of cases was that of Koop, Jordan, and Horn [2] in 1947. They reported 5 instances of neurilemmoma of the pharynx, bringing to 11 the total number so far recorded. In 1949 Slaughter and de Peyster added four cases. Since then only sporadic reports of single cases have appeared in the literature.* Guggenheim [7] recorded a case in 1953 and after carefully reviewing the American and foreign literature was able to tabulate a total of 36 cases. Couch [8] added another case, in 1954.

The infrequency of this tumor and the fact that it represents the first one ever reported in a Puerto Rican prompted us to report the case.

REPORT OF CASE

In December, 1954, a 34-year-old Puerto Rican mulatto was seen for the chief complaint of dysphagia for solids and the feeling of a growing mass in the right side of the throat. He complained of profuse sialorrhea and of slight pain radiating to the region of the right ear. No change in his voice and no other neurologic signs or symptoms were noted. The symptoms started about six months prior

* References 4 to 6.

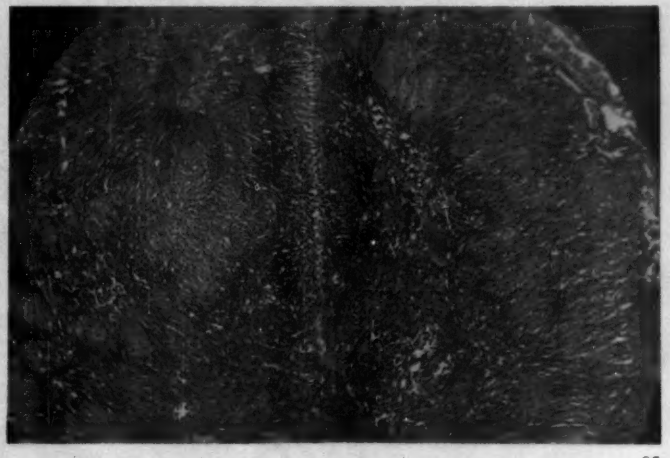

Fig. 1.—Closely packed Schwann sheath cells in the Antoni A type of tissue; hematoxylin and eosin; reduced ¼ from mag. × 80.

Fig. 2.—Palisaded arrangement of Schwann cells forming Verocay bodies; hematoxylin and eosin; reduced ¼ from mag. × 360.

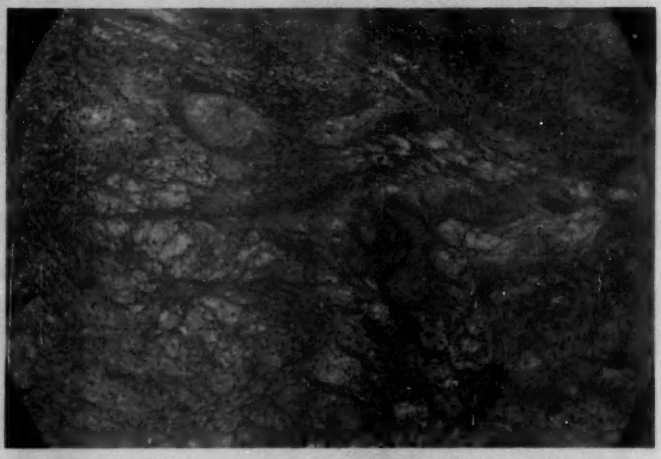

Fig. 3.—Loosely edematous and vacuolated Antoni B type of tissue; hematoxylin and eosin; reduced ¼ from mag. × 80.

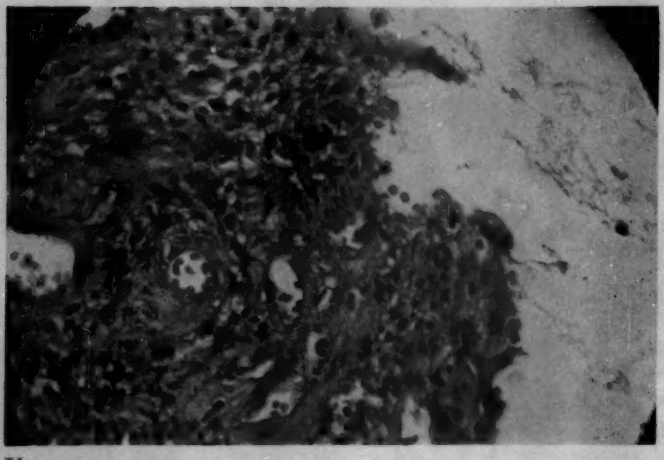

Fig. 4.—Blood vessels with prominent collagen sheaths around them. A large zone of cystic degeneration is evident at the left. Hematoxylin and eosin; reduced ¼ from mag. × 360.

to admission but had progressed markedly over the last two months.

Examination revealed a large ovoid mass in the right side of the pharynx, the sagittal and longest diameter of which extended from slightly above the level of the soft palate to a level equal to that of the base of the epiglottis. The mass was firm, nontender, and covered by intact mucosa. The cervical lymph nodes were not enlarged. No other pertinent findings were noted in the physical examination.

The tumor was completely removed through an intraoral approach. The patient's recovery was uneventful. At present he only complains of pain around the right eye and ear. No neurologic deficit was noted.

The neoplasm measured 5 by 3 cm. and was partially covered by normal mucosa. On section it was well encapsulated and composed of firm and grayish-white fibrillary areas separated from each other by loose and edematous tissue with occasional small cystic spaces. Microscopically these alternating zones corresponded to the classical arrangement in neurilemmomas of Antoni Types A and B tissues. The former is composed of compact groups of Schwann cells, with palisading of nuclei and formation of Verocay bodies. The nerve sheath cells are loosely arranged in an edematous matrix, with cystic degeneration in the Antoni Type B tissue. Blood vessels are numerous, and many of them are surrounded by thick collagen sheaths.

COMMENT

Benign encapsulated neurilemoma was the name given by Stout[9] in 1935 for this tumor of peripheral nerve sheaths described by Verocay[10] in 1908. The name very well implies its benign nature despite the fact that in many of them anaplasia may be a prominent feature. The tumor is not frequent in the head and neck. Only 38 cases, including the present one, have been reported in which the tumor developed in the pharynx.

Half of the cases have developed in persons below the age of 40 years, about the same number occurring in males.

The most frequently encountered symptoms are those of dysphagia, changes in phonation, sensation of a mass, and pain, either upon swallowing or radiated to the neck or ear. Many of these symptoms are due to involvement of the cervical sympathetic or any of the cranial nerves at the pharynx. In the great majority of cases, as the tumor originates from the involved nerve, the neurologic deficit persists or is aggravated after surgery.

The tumor should be considered as a malignant one on account of its position, and for this reason we agree with those who recommend surgical removal. The lesion is definitely radioresistant. The surgical approach is more frequently an intraoral one, but a cervical and even a combined cervicopharyngeal type have been used.

SUMMARY

A case of neurilemmoma of the pharynx is presented. It represents the first one ever recorded in a Puerto Rican.

A complete review of the literature on the subject is attempted.

The more important clinical and pathologic high lights of the tumor are enumerated.

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Clinical Notes, New Instruments and Techniques

ACRYLIC SEAL

JACOB SCHAFFER, D.D.S., Newark, N. J.

I should like to make known to otologists a successful method of protecting ears, either with perforated drums or with healed postoperative cavities, from the entry of water in swimming. A cast of the outer auricle and external portion of the canal is made in the manner of a flexible plastic covering, using the technique followed in making dentures.

Fig. 1.—Acrylic seal in place in the ear.

Fig. 2.—Internal view of acrylic seal. Note how it fits into all undercuts.

An impression of the entire ear is taken with a colloid material. This material, being elastic, will take an accurate impression of all the undercuts in the auricle as well as the outer portion of the external auditory canal. A model is then poured and a semisoft acrylic cast is formed. When finished, this will lock into place and be a self-retentive complete seal. It has been used satisfactorily by a number of postoperative cases, including a physician.

31 Lincoln Park (2).

Submitted for publication Aug. 15, 1955.

Society Transactions

AMERICAN LARYNGOLOGICAL ASSOCIATION

Henry B. Orton, M.D., President

Harry P. Schenck, M.D., Secretary

Abstracted by Samuel Salinger, M.D.

Seventy-Sixth Annual Meeting, Hollywood Beach, Fla., March 13-14, 1955

The Indications and Technique of Lymph Node Dissection of the Neck. Dr. Alton Ochsner.

Metastasis to the regional lymph nodes is very common with malignant lesions about the oral cavity because of the rich lymphatic drainage. Most of these remain localized in the cervical nodes for a long time except in the case of transitional-cell carcinomas and lymphoepithelioma, which may metastasize widely very early. The decision to do a neck resection may at times be difficult to make, because frequently enlargement of the glands may be due solely to inflammatory causes and, on the other hand, metastases may be present without palpable enlargement of the glands. This accounts for the divergence of opinion among surgeons as to the feasibility of prophylactic resection. According to Dr. Ochsner, the procedure is not necessary in cancer of the lip unless the lesion is greater than 3 cm. in size or is higly malignant. Metastases to the glands are more frequent in lesions of the upper lip than of the lower. In large lesions or anaplastic lesions without apparent gland involvement, a unilateral suprahyoid dissection is indicated if the lesion is lateral; if in the midline, a bilateral resecton would be necessary. In the presence of suprahyoid involvement, a unilateral neck dissection is done or a bilateral dissection at different stages if the lesion is near the midline. Neck dissection in connection with carcinoma of the tongue or floor of the mouth may require a suprahyoid or unilateral neck dissection or even a bilateral resection en bloc with the mandible, depending on the location, extent, and type of the primary lesion.

In no case is a neck dissection to be done unless the primary lesion is completely eradicated.

DISCUSSION

Dr. LeRoy A. Schall inquired as to the complications following resection of the carotid artery and vagus nerve in the complicated cases. He had had five such cases in recent months.

Dr. Henry Orton asked the essayist for his opinion regarding papillary cyst adenocarcinomas of the thyroid, since there has been some difference of opinion as to proper therapy.

Dr. Ochsner, in closing, replied that when it is necessary to resect the carotid artery they do so without fear of consequences because of the availability of grafts which can be immediately utilized. Resection of the vagus is a cheap price to pay for the possibility of a cure. Concerning the cystadenomas, he feels that they are truly lymph node metastases from carcinoma of the thyroid rather than aberrant thyroids and should be dealt with by radical excision, frequently bilateral. If the lesion is unilateral it is frequently impossible to palpate the original tumor in the thyroid. In such cases he does a hemithyroidectomy, including the isthmus. If a tumor is found in the thyroid they do nothing more than keep the opposite side under observation for a long time. If no tumor is found they remove the other half of the thyroid and do a bilateral lymph node dissection. He feels that the earlier the lesion is discovered the more radical should be the resection, since this is the best opportunity to obtain a cure.

Evaluation of Neck Dissection in Carcinoma of the Larynx. Dr. Louis H. Clerf.

In a series of 258 total laryngectomies Clerf found that metastases to the regional lymphatics occurred in 25%-30% of the cases. The five-year survival rate in 34 cases where neck dissection was carried out was 26.4%. Of the 33 not treated surgically but with irradiation only, 1 survived five years, so that of the total 67 cases with metastases only 15% survived beyond five years. Since 1940 he has been doing a neck dissection combined with the laryngectomy in a greater

number of cases than formerly, despite the absence of palpable lymph nodes, with a much higher percentage of survivals. He points out that the cases belonging to this category are those in which the lesion is supraglottic or infraglottic, or where the arytenoid is involved and the mobility of the cord is definitely impaired. Analysis of the group by five-year periods shows that cases where elective resection was done concurrently with the laryngectomy yield a much higher percentage of recoveries than those where a secondary resection was performed.

DISCUSSION

DR. CHEVALIER L. JACKSON said he had every intention of discussing the paper but found after hearing it that he was in complete agreement with the author and could find nothing significant to add to it.

DR. JULIUS W. MCCALL inquired about complications following bilateral resection of the jugular veins. He had seen three such cases in which disastrous brain sequelae had been encountered.

DR. EDWIN N. BROYLES was somewhat dubious about the necessity for gland resection in cases of carcinoma of the larynx where the tumor was entirely intrinsic. He had done resections in several cases without finding any metastases in the glands. On the other hand, in cases where the peritracheal glands are palpable it is likely that the thyroid is also involved, and, of course, in such cases radical dissection is indicated.

DR. H. H. HARRIS (Houston, Texas) inquired whether Dr. Clerf removes the thyroid gland in toto or only the half that appears to be involved.

DR. ANDREW EGGSTON, as a pathologist, brought up the point that frequently it is difficult to appraise the tissue submitted because the pathologist has no idea of the exact situation of the suspected mode in the living state. It would take days to go through the mass section by section to determine the exact location and extent of the metastasis, since frequently it is limited to individual cells. The surgeon who removes the tissue has a much better idea of its relationship to the original focus than has the pathologist. Also a confusing factor is the presence of hemorrhagic foci indicating a noditis, which might be mistaken for a metastasis, thus the difficulty in offering a prognosis based merely on the tissue examination.

DR. JEROME A. HILGER called attention to the complete absence of mortality in the series presented, which speaks volumes for the excellent technique and postoperative care. Increased interest in this type of surgery in recent years brings up the question as to the proper delegation of these cases. Many men have reached the age when they do not care to expand into new techniques. It points to the necessity for proper leadership and the expansion of educational growth among those who are still growing up in the specialty.

DR. CLERF, in closing, pointed out that they did have mortality but not in the selective group. The point is that elective block dissection in conjunction with laryngectomy, when indicated, does not enhance the mortality to any great extent.

Bilateral dissections are fraught with some danger. One case that survived complained of dizziness, had a florid complexion and marked scleral injection. He ultimately died of metastases below the clavicle. Certainly one cannot be too enthusiastic about bilateral block dissection, yet, as Dr. Ochsner stated, it is not too great a price to pay if there is a chance to save a life.

As to Dr. Broyles' comment, Dr. Clerf feels that if the patient has an extensive lesion, even though there are no palpable metastases, the chances for a permanent cure are much better if a unilateral block dissection is carried out, since the added risk is minimal. This attitude is prompted by the experience of having patients previously laryngectomized return with inoperable nodes in the neck. Concerning metastasis to the thyroid, the surgeon is definitely behind the eight ball. The operative mortality is increased and the operative procedure prolonged. Certainly the prognosis in such cases is not favorable. Concerning Dr. Eggston's remarks, Dr. Clerf, far from appearing critical, expressed his deep appreciation for the contribution of the pathologist, realizing the great difficulty of accurate and thorough study of all sections of the mass submitted, a matter that is limited by the shortage of personnel as well as by time limitations in most hospitals.

Mixed Tumors of the Posterior Pharyngeal Wall. DR. FRED Z. HAVENS and DR. L. C. BUTLER JR.

Mixed tumors of the posterior pharyngeal wall are very rare. Havens and Butler were able to find only 41 cases over a period of 37 years at the Mayo Clinic. The majority occurred

during middle life. In 11 cases where the postoperative period was less than five years there was one recurrence. In the 30 cases in which all but 1 were followed more than five years there were 24 with no recurrence and 5 with recurrence (3 of which were successfully treated and 2 succumbed to the disease). Most of the patients were operated on via the transoral route after preliminary ligation of the external carotid artery, the mass-including capsule being scooped out and the cavity left open with only an iodoform gauze pack. Several cases were operated on via an external approach through the carotid area with primary closure. They believe that the danger of seeding is greater when the latter procedure is employed because of the closure of the cavity operated on.

DISCUSSION

DR. HENRY B. ORTON mentioned a case seen recently in which it was necessary to remove a portion of the ascending ramus of the mandible because of the extent of the growth. It was not necessary to ligate the carotid, and the approach afforded much more room than would have been available via the intraoral route.

DR. HARVEY P. SCHENCK stated that in the few cases in which he had seen these growths they were readily removed by the transoral route. One case was interesting because it had been treated for some time as a peritonsillar abscess. One of the dangers in removing these growths arises out of the fact that the normal mucosa overlying the mass has to be incised to reach the capsule, which is attended by the risk of seeding and subsequent recurrence. Hemorrhage in his experience has not been a disturbing factor. After removal, one may be astounded by the depth of the resulting cavity. Dr. Schenck has practiced suturing from the bottom up and obtained very satisfactory cosmetic results.

DR. ALBERT C. FURSTENBERG emphasized the importance of thorough eradication at the first attempt because recurrences are usually found in areas remote from the original focus, increasing the difficulty of removal. Besides, the recurring growth is usually more malignant than the original.

He agrees that seeding may be a very potent factor in the incidence of recurrences. It is therefore advisable, particularly if the mass is in one of the salivary glands, to remove some of the tissue surrounding the capsule of the tumor even though it may appear grossly benign.

DR. DEGRAF WOODMAN asked Dr. Havens' recommendations as to biopsies in view of the danger of seeding.

DR. HAVENS closed the discussion by stating that he had at times been tempted to attack the tumor via the external route, as suggested by Dr. Orton, but had thus far not encountered a case when this procedure was imperative.

As for ligating the carotid, it depends on how readily one is alarmed, which was the case in his own experience on several occasions. He would therefore feel more secure if he ligated. The matter of closing the defect is one that may be left to the individual surgeon. Dr. Havens does not suture but fills the cavity with iodoform packing, which is removed gradually, beginning on the fourth day. The wound heals readily, and at a later date one can barely see the area operated on. He believes also that there is less danger of seeding.

Dr. Havens agreed heartily with Dr. Furstenberg on the matter of adequate surgery at the first attempt. This applies to all malignant tumors as well. Concerning the question of biopsies, he is in the habit of sending a biopsy specimen out after the capsule has been identified and the dissection partly completed. As a matter of fact, it makes little difference, since the excision is going to be wide enough to encompass all the presenting mass.

Vasomotor Rhinitis. DR. THEODORE E. WALSH.

Walsh has been unable to identify allergic factors in a large percentage of the cases of vasomotor rhinitis which he has observed. In most cases the etiology is to be found in endocrine, dietary, and psychosomatic disorders. Hypothyroidism, often of the subclinical level, is very common, and good results may be expected following the administration of thyroid extract in adequate doses. In children dietary indiscretion, such as the ingestion of excessive amounts of carbohydrates, is frequently the cause. Emotional states should not be overlooked, because in many instances they act as a trigger mechanism that sets off allergic manifestations in many persons who are only mildly allergic without showing specific sensitivity.

Walsh decries the continuous use of vasoconstrictors, which only leads to subsequent turgescence. He advises phenylpropanolamine (Propadrine) by mouth for temporary relief in addition to the care of such constitutional factors as have been mentioned.

DISCUSSION

DR. OLIVER E. VAN ALYEA expressed his opinion that the condition described by Dr. Walsh was a borderline or atypical allergy and might be considered as a separate entity. A question he should like to raise apropos of therapy relates to the employment of cauterization of the turbinates in stubborn cases. This is an old-time procedure, of which he does not approve.

DR. HENRY L. WILLIAMS agreed that many of these cases are difficult to relieve. It occurred to him that it might be possible to differentiate among them a little more accurately. In his experience the cases with violaceous mucous membranes are usually the so-called low B. M. R. without myxedema, which can successfully be treated. On the other hand, the cases with watery discharge and relatively normal mucosa are not helped by endocrines and are better classified as physical allergy. The insistence of Dr. Walsh on antigen-antibody reactions is not in line with the thinking of immunologists. The concept of physical allergy cannot be disproved, and allergists are willing to admit that many of these cases which are clinically indistinguishable from true allergy do not yield a positive skin test. The dietary regimen mentioned by Dr. Walsh is reminiscent of Dr. Sam Roberts' work along these lines, which has proved to have great merit. As for cauterization, mentioned by Dr. Van Alyea, Dr. Williams has found it efficacious, particularly if applied in the region of the sphenopalatine ganglion. He has seen no ill effects from the procedure, there being no permanent damage to the cilia, and it affords relief for long periods of time.

DR. LOWENTHAL called attention to possible emotional or psychiatric influences in these cases. Frequently the patient will give a history of spastic colitis or some other derangement of the sympathetic nervous system. A pseudopsychiatric approach may in many instances afford the key to a solution of the problem.

DR. WALSH, in closing, agreed that cauterization of the ganglion is free from harm and is not to be confused with cauterizing the turbinal, which may alter the physiology of the mucosa. He personally likes injecting the ganglion, which has given him some very good results. Concerning physical allergy, Dr. Walsh felt incompetent to discuss it adequately, although he recognizes it as an entity. However, these cases are a minority in the group he was discussing. He agrees that the psychosomatic approach has much to offer yet does not mean that all these patients must consult a psychiatrist. The solution to the problem lies in a thorough understanding of the patient as a whole, knowing his background, habits, environment, and emotional status. Frequently the trouble is due to a combination of factors aside from the purely allergic.

A Few High Lights Concerning Respiratory Cilia. DR. G. EDWARD TREMBLE.

The electron microscope has in recent years proved of great value in determining the structure of cilia. "Each cilium consists of a basal body which is imbedded in the cell. Just above the basal body a small cuticular ring can often be seen." The rings of adjoining cells seem to be attached to each other, forming a thin plate. "The cilium itself consists of an axial fibrillar bundle and a semitransparent, thin peripheral sheath."

Much of the experimental work in animals is described in the article.

It is assumed by some that the activating agent which produces the ciliary beat arises in the basal bodies and is transmitted through the longitudinal fibrils to the tip of the cilia. The question as to the exact origin of the cilia is still undetermined; we do not know whether they grow from the surface of the cell or develop below in the basal body. Also still a mystery is the exciting influence which causes the ciliary beat.

DISCUSSION

DR. AUGUST L. BECK recalled an early motion picture produced by the late Dr. Mosher in which one cell had become detached and was still showing activity, the cilia continuing to beat in rhythm with the rest of the cilia along the main body of cells. It reminded him of a soloist and orchestra in action though some unseen agency.

DR. ARTHUR W. PROETZ called attention to the value of the electron microscope, which has in recent years vastly increased our knowledge of ciliary activity. The time now is ripe for some genius to unravel the mystery of the true origin and nature of ciliary activity. It requires not only the electron microscope but an observer with imagination who can put together such facts as Dr. Tremble presented along with other data. The situation is almost analogous to the study of the circulatory system, in which all the facts were known for years but not clearly understood until someone discovered valves which finally proved that the blood was going in one direction.

DR. TREMBLE closed the discussion by acknowledging Dr. Beck's comment about the continual activity of cilia even when the cell is detached. This observation of Dr. Mosher's has been corroborated many times since. By having a patient with a cold blow his nose onto some wax paper and placing the secretion under the microscope, one may frequently see detached cells with cilia still waving. They will continue to wave as long as attached to protoplasm, kept warm and moist. His object in bringing this subject before the Association was to emphasize the constancy of cilia throughout the animal kingdom, as shown by comparison with spermatozoa with its fringe-like tail and the nine-paired groups around the periphery with the two larger central ones, just like the cilia in our own nasal mucosa.

The Radical Obliterative Frontal Sinus Operation: A Consideration of Technical Factors in Difficult Cases. DR. ROBERT L. GOODALE.

Dr. Goodale proposes a radical obliterative operation for cases in which the Jansen-Lynch-Smith procedure is inadequate, cases of extensive trauma, chronic osteomyelitis, and recurrences following previous surgery. The previous objection to this method, namely, the resulting deformity, can be overcome with modern plastic procedures. The obliterating operation must be complete, all mucosa and bony septa removed, the duct destroyed, and the ethmoids exenterated. The posterior wall of the sinus is not removed unless apparently diseased or where there is a suspicion of some intracranial extension. Eight cases are shown in which the excellent plastic repair of the defect demonstrates the fine cosmetic results obtained. In most cases autogenous bone grafts were employed.

DISCUSSION

DR. JAMES M. ROBB said that Dr. Goodale's paper has shown definitely that it is an erroneous concept to conclude that the sulfonamides and antibiotics have completely done away with the necessity of operating on the frontal sinus.

DR. W. LIKELY SIMPSON felt that, while 100% perfect postoperative results were impossible, the usual course after a proper procedure should be as good as that following a radical mastoid. Such factors as the extent of the disease of the bone and membrane, as well as the size of the sinus, have a definite bearing on the postoperative course. It is imperative that all of the mucous membrane be removed and the entire ethmoid area exenterated. As for osteomyelitis, it is better to defer the definitive surgery until the acute process has been brought under control. Through the usual brow incision the exenteration can be accomplished, starting from the upper extension of the sinus and working downward. The polishing burr is a useful adjunct in the finishing process. Where the sinus extends far upward it is advantageous to make the incision at the supreme extremity, which affords good access and which leaves very little scar or deformity. Cases are frequently presenting themselves which require the type of surgery described by the essayist, and the individual surgeon should have the courage to go ahead when the case demands it.

DR. FRANCIS W. DAVISON recalled a technique described by Dr. Jesberg a few years ago, in which the floor of the sinus is removed along with the outer third or half of the anterior wall, a procedure which is indicated and has been proved successful in the large or expanded sinus.

DR. JAMES MAXWELL stated that in recent years they have attacked the large frontal sinuses via a coronal incision, which permits excellent exposure of the entire frontal bone and, if the orbital ridges are removed, affords access to all the ethmoids and sphenoids. He asked Dr. Goodale whether with the brow and vertical incisions it was his practice to pack the wound wide open.

DR. GOODALE closed the discussion by first agreeing with Dr. Robb that too much reliance has been placed on antibiotics by some surgeons, particularly in chronic cases, gross infections, etc. Besides, antibiotic therapy needs reviewing in the light of the emergence of resistant strains and new bacterial invaders. Certainly the pyocyaneous and resistant staphylococcic infections call for more effective medicaments than at present are available. He was interested in Dr. Simpson's use of the burr. Having been trained in the use of gouge and rongeur, he had clung to them because of his familiarity with them. Nevertheless, the idea of using the burr, particularly from above, seems appealing. The important point is to see that the utmost limits of the sinus are reached.

He recalls one case of a chronic frontal sinusitis with a localized osteomyelitic process in which the Jesberg procedure worked out very well, although some deformity did result.

He agreed with Dr. Maxwell that the coronal incision can afford a wide exposure. However, in the cases he has presented it seemed more feasible to go through the incisions of the previous operations rather than make new ones. In the one case in which a vertical incision was used, it was necessitated by the fact that after the Lynch exposure it was found that an osteomyelitic process was present not previously seen in the x-rays.

Rehabilitation of Patients with Clefts of the Lip and Palate (Motion Picture). DR. DEAN LIERLE. (No formal paper.) (No discussion.)

Unilateral Cord Paralysis. DR. DANIEL S. CUNNING.

Dr. Cunning studied a series of cases of vocal cord paralysis observed at the Manhattan Eye, Ear and Throat Hospital over a period of 15 years. There were 163 males and 99 females; 82 involved the right cord and 180 the left. The etiologic factor was found in 68.7% of the group, the remainder being unidentified. Neoplastic and toxic or inflammatory lesions accounted for almost half the cases.

A search for the etiologic factor must include a thorough physical, x-ray, neurologic, and serologic examination. The causes are too numerous to mention, since any lesion from the nuclear and supranuclear areas in the brain and on down along the course of the vagus may affect the functioning of the nerve, which may also be involved in any number of general constitutional disorders of a toxic nature.

DISCUSSION

DR. LOUIS H. CLERF recalled a statement made by Dr. Kyle years ago that the general physician sees this condition from the systemic standpoint, while the specialist looks at it as a local affair, which he often treats without realizing its significance. Dr. Cunning's statistics agree with previous studies, namely, that neoplasms are the cause in two-thirds of the cases, the others being considered idiopathic. While there is not much to be done for the neoplastic group, the laryngologist can be of great assistance to the thoracic surgeon who realizes that left-sided paralysis due to thoracic lesions rarely is amenable to surgery. Dr. Gibbon, who has explored many of these cases, has yet to find one which he could successfully extirpate. Another interesting item is the occurrence of a left-side paralysis 5 to 10 years after a mastectomy, indicating an inoperable mediastinal extension. As to post-thyroidectomy paralysis, it indicates the importance of the surgeon learning more about the recurrent nerve. The toxic group of cases is interesting, since many of the idiopathic cases are of this nature, being probably due to a virus. These cases offer a better prognosis than many of the others. Dr. Clerf agrees with Dr. Cunning as to the value of speech therapy in some of these cases. Surgery such as that recommended by Dr. Morrison can be successful in improving the voice when the cord is abducted with poor tension.

DR. EDWIN N. BROYLES mentioned a case of spontaneous unilateral paralysis in which the examination offered no clue as to the cause until eight months later, when a mass developed in the thyroid region which proved to be malignant. In another case of unilateral paralysis it was a year before a tumor of the mediastinum was diagnosed. It might be feasible in such cases to explore the left recurrent nerve and follow it down into the chest in the hope of finding the lesion. It is his opinion, as well as that of surgeons of his acquaintance, the tumors of the thyroid causing a recurrent paralysis are usually malignant. As for patients being improved by speech therapy, this may be true of the speaking voice but is usually ineffective insofar as the singing voice is concerned.

DR. C. L. JACKSON reported having seen two cases on successive days in which the etiology could not be determined. He inquired whether Dr. Cunning had had any results from medical therapy in the toxic or inflammatory cases.

Recurrent paralysis following diverticulectomy had disturbed him in several cases, and he wondered whether the essayist had anything to offer by way of preventing this complication.

DR. JULIUS W. MCCALL thought that unilateral paralysis caused a voice weakness rather than a true hoarseness, because he has observed that these patients have a better voice in the morning than later in the day.

DR. BENJAMIN SHUSTER mentioned a case in which neurologic findings pointed to a brain tumor. However, a more careful study of cranial nerve function and the reaction to skin stimuli revealed a loss of sensation on one side, pointing to a spinal thalamic involvement on the side opposite the cord paralysis.

DR. FRANCIS L. WEILLE asked Dr. Cunning whether he attached any significance to the anastomotic branch from the superior to the inferior laryngeal nerve insofar as explaining the position of the cord after injury.

DR. CUNNING closed the discussion first by evaluating what he called hoarseness. The character of the voice in larynx lesions often varies with the lesion within wide limits. So far as he is concerned, he can only call it hoarseness, though in some cases it may be partly fatigue.

It is true about 10% of the cases are caused by cerebral or spinal lesions, and it is therefore always important to check all the reflexes for possible signs pointing to the site of the lesion. As for medical treatment, they have used iodides, vitamins, and cortisone, all with no effect. There were no cases of paresis complicating diverticulotomy in his series.

There is no question that the laryngologist is up against it in so many cases because of the difficulty in discovering the etiology. They often go for years with no other symptom than the hoarseness.

Ten-Year Progress of Laryngofissure Operation. DR. MERCER G. LYNCH.

Experience with the laryngofissure operation in recent years has yielded a higher percentage of cures: up to 90%. This has been accomplished through wider excision of tissues. Where the commissure has been involved a portion of the cartilage is removed and the excision extended into the opposite cord. The possibility of a web resulting is not viewed with any alarm, since the McNaught procedure with the tantalum plate has proved its worth. Dr. Lynch does not recommend it being done at the primary operation because of the granulation tissue which springs up in abundance from the irritation of the tissues by the metal. It is better in these cases to await scar formation before attempting to insert the tantalum plate. Laryngofissure can be successful even in cases where the growth has extended as far as the vocal process and even when mobility of the cord is limited. However, if the arytenoid itself is involved or mobility is definitely limited, one would have to choose laryngectomy in preference. It is important to resect well beyond the apparent lesion, and Dr. Lynch recommends following the surgery with the application of electrodesiccation. Also, he calls attention to the fact that tracheotomy is not resorted to, which materially reduces the postoperative period.

DISCUSSION

DR. CHEVALIER L. JACKSON was glad that Dr. Lynch had raised his voice in favor of the laryngofissure operation. The indications for it have broadened rather than contracted because of broadened techniques. The elder Jackson's technique of removing anterior commissure lesions has been improved since Broyles described the anterior commissure tendon, so that now, instead of cutting the cartilage in the midline, the incision goes through the healthy side, thus permitting excision of the midline cartilage along with the lesion. Regarding web formation following thyrotomy, he has found the procedure suggested by Dr. Gabriel Tucker of value, namely, anchoring the epiglottis forward with a suture in all cases where its attachments have been severed.

Also, Dr. Jackson pointed out that this operation should really be termed a partial laryngectomy rather than a laryngofissure.

DR. HENRY M. SCHEER called attention to a procedure described by his son, in which the laryngofissure approach was employed for the relief of a bilateral abductor paralysis. One paralyzed cord was tied back with a wire suture, and the patient for three years was still breathing comfortably through the larynx.

DR. LYNCH, in closing, stated that he does not favor a hemilaryngectomy in cases where the midline incision is not indicated. He would prefer laryngectomy in such instances. With regard to web formation, he has not employed Dr. Tucker's procedure but has used the tantalum splint, with good results. As for Dr. Scheer's operation, Dr. Lynch recalled the submucous dissection of one cord reported by Dr. Hook of Boston in which some good results were obtained.

General Tonsillectomy: Suggestions for Management and Peritonsillar Injections. DR. PAUL M. MOORE.

Moore reviewed the subject of tonsillectomy under general anesthesia, laying emphasis on the value of injecting the peritonsillar area with a saline solution containing epinephrine, 6 minims to the ounce, which not only facilitates the dissection but also markedly reduces the bleeding. He

described the preoperative management of the child with the object of minimizing psychic shock. Up to the age of 14 atropine is administered in adequate dosage; older persons are given a barbiturate. Efocaine for the reduction of postoperative pain has proved hazardous and has been discarded. He has found that the preoperative use of cortisone or hydrocortisone is of value in the "thymicolymphatic" type of child. Also, the intravenous administration of hydrocortisone has been shown to be effective in combating the hypotension of surgical shock. The operation is performed in the head-low position, using the Davis gag with attached tongue blade.

DISCUSSION

DR. THOMAS C. GALLOWAY cited statistics from the Evanston Hospital showing a decrease in tonsillectomies from 1000 in 1945 to 400 in 1953. This reveals a trend to reevaluation of the indications. Regarding the relationship of tonsillectomy to the incidence of poliomyelitis, he feels that, despite conflicting theories tending mostly to show a predisposition on the part of the former to the latter, the theory of a barrier being removed is not compatible with present theories of the pathogenesis of poliomyelitis. X-ray irradiation is again beginning to gain favor if properly administered by an experienced roentgenologist. Rhodes' recent paper showing the incidence of bacteremia following tonsillectomy is very impressive, particularly since it revealed a lower incidence in cases done by the Sluder or Beck method, the incidence being proportional to the time consumed in the operation.

DR. HARKINS reported a lowered incidence of bleeding during and after surgery, which he attributes to the routine use of vitamins K and C before, during, and following the operation. Also, he believes good results can be obtained from the use of Adrenosem, 5 mg. IM, immediately after the operation.

DR. MOORE, in closing, commented on an editorial in the *Journal of the American Medical Association* which stated that a tonsillectomy performed at any previous period of time rendered the person more susceptible to poliomyelitis. This seems to overlook the simple fact that surgical repair or normal healing, far from removing a normal barrier to infection, offers a natural obstacle. Regarding the use of the vitamins, he is convinced that they are of great value. Certainly children receiving aspirin will show a low prothrombin. As for Adrenosem, his experience with it in a few cases of nasal hemorrhage has not been convincing. He has not tried it in the tonsil cases.

Carcinoma of Maxillary Sinus in Youth. DR. BERNARD J. MACMAHON.

Because it is very unusual to encounter a carcinoma of the antrum at the age of 20, Dr. MacMahon presented such a case along with a discussion of the therapy. The first symptom was numbness of the cheek, followed by swelling and some displacement of the bulb. X-rays indicated pathology in the antrum and ethmoids, and the sinus was opened on the assumption that a suppurative process was present with involvement of the orbit. However, the histologic examination of tissue removed showed a carcinoma which appeared to be of the radiosensitive type. Surgery was not advised because of the extent of the growth, and the patient was given irradiation therapy. There was considerable improvement locally, but metastases, first in the neck and then more widespread, proved to be beyond the hope of a cure by any means, and the patient died in a very short time. Dr. MacMahon cited Professor Ohngren to the effect that the general impression that metastases occur later is erroneous. The rich network of lymphatics in the mucosa of the antrum communicates freely with the larger trunks in the neck leading to the glands.

DISCUSSION

DR. ANDREW EGGSTON has noted changes in the classification of tumors based on studies of histogenesis since World War II. The slides shown by Dr. MacMahon are most likely squamous-cell types. The anaplastic cells take on peculiar behavior patterns, often undergoing metaplasia. This accounts for differences of opinion as to nomenclature, depending on who sees the slide and what portion of it impresses them. Apparently more malignancies are being observed in younger persons than formerly. Recently a case in a child aged 5 years, originally diagnosed as fibrosarcoma of the sphenoid or ethmoid, was later pronounced a neuroblastoma when studied by several pathologists. Despite radiation therapy the child developed metastases and died. Two other cases of neuroblastoma were seen by Dr. Eggston: one in a boy of 12 and the other in a woman of 45. Whether this is a new type of tumor or a reclassification of an old one is

not certain. Yet the fact remains that they are anaplastic and highly malignant. Another problem that is perplexing is the increased incidence of so-called papillary sinusitis. These were thought to be akin to recurring papillomata of the larynx, often attributed to a virus. Yet in some cases after a long time a recurrence will appear with every indication of malignancy.

DR. FREDERICK T. HILL also has recognized increasing frequency of malignancy in the young. He has been impressed by the symptom of numbness of the face, which is highly significant in early diagnosis. He inquired whether Dr. MacMahon had found x-ray evidence of involvement of the canine convexity or orbital plate. Frequently stereoscopic views will bring out early changes.

DR. LEROY A. SCHALL agreed that the symptom of numbness of the cheek is a very important one. Other symptoms which have been observed are unilateral nasal obstruction, recurring hemorrhage, exophthalmos, pain in the teeth, and impaired vision. An analysis of 212 cases by the speaker revealed the majority to be carcinomas, yet only 2 were in children, although rhabdomyosarcomas and other types of sarcoma were also found. Dr. Schall recalled several other cases in which the symptoms mentioned above were helpful in the diagnosis and supported by good x-ray pictures. The essence of the thing is that one should always be cancer-conscious and study these cases carefully in their early stages.

DR. C. E. MUNOZ-MACCORMICK (Porto Rico) reported a case in a child of 4 years with pain and swelling of the cheek where the x-rays showed merely a ballooning of the walls of the sinus. Surgical exposure revealed a highly malignant mass which had eroded the bony walls even into the pterygomaxillary fossa.

DR. PIERCE THEOBALD reported a case in a 48-year-old enlisted man, whose early symptom was nasal obstruction. It was treated as a sinusitis for some time before a mass appeared in the nose, which on biopsy revealed an anaplastic malignancy. X-rays showed extensive involvement of the sinuses, which was confirmed by surgery. Apparently it was too widspread to be completely removed, and the outcome was inevitable.

DR. MACMAHON, in closing, stated that the x-rays were carefully studied in his cases and the contour of the canine fossa and orbital plate appeared unchanged.

The symptoms mentioned by Dr. Schall are all very important, and he agreed that numbness of the cheek is particularly significant.

Functional Disturbances of the Upper Swallowing Mechanism. DR. JOHN R. LINDSAY.

Functional disturbances of the swallowing mechanism may be divided into three groups: those affecting musculature, innervation, and psychic control. Among the first named are such conditions as myasthenia gravis, generalized scleroderma, dermatomyositis, and systemic conditions such as tuberculosis, thyrotoxicosis, and senility. These may be demonstrated in the barium test when the fluid tends to remain in the valleculae after swallowing. Neurologic conditions affecting the swallowing function comprise peripheral neuritis, jugular foramen syndrome, intracranial lesions affecting the nerves, and lesions pressing on the bulb. The cricopharyngeus may retain its function in some cases or may lose some of its efficiency in others. Examination during the Valsalva maneuver will often determine the efficiency of this muscle. Disturbances of psychic origin are of manifold origin and are commonly found in some types of mental illness. Organic disease must be excluded by careful examination before such cases are turned over to the psychiatrist.

DISCUSSION

DR. CLAUDE C. CODY noted the reference to age as a factor. He believes that deficiency in vitamin B_2 is frequently observed in the aged. It has a bearing on the motility of the esophageal musculature through its action on the innervation. This has been noted also in connection with musculature weakness in tuberculosis. He inquired whether Dr. Lindsay had seen any evidence of vitamin B_2 deficiency.

DR. WALTER B. HOOVER has seen cases of bilateral recurrent nerve paralysis but has failed to observe involvement of the cricopharyngeus, which he believes is not innervated by the recurrent nerve. Also, he has observed cases of 10th-nerve paralysis in which the pharynx and the vocal cord on one or both sides were paralyzed and in which the cricopharyngeus was markedly weakened or relaxed. He should like to know Dr. Lindsay's ideas on the innervation of the cricopharyngeus from the pharyngeal plexus and whether a lesion of the upper portion

of the nerve near the jugular formen or intracranially would involve this muscle. He suggests that in cases where x-rays fail to demonstrate relaxation of the cricopharyngeus one might well esophagoscope the patient to rule out a possible fibrosis or web formation as the cause of the dysphagia.

Dr. Edwin N. Broyles recalled three cases of more or less mechanical difficulties in swallowing in the region of the cricopharyngeus. One case which had been diagnosed as hysterical globus was due to a web formation, and the other two revealed definite fibrosis, which rendered passage of the scope very difficult even under general anesthesia.

Dr. S. C. Missal reported having seen a number of cases of poliomyelitis with marked difficulty in swallowing associated with a change in the voice. Yet the laryngeal function was good and the palate muscles seemed to act normally. He wondered whether this alteration of the voice was not due to inadequate control of the air column by the pharyngeal sphincter.

Dr. Lindsay replied to Dr. Cody's question by stating that the case in which the x-rays were shown did not have a vitamin deficiency. The patient had to be tube fed because of pain due to ulceration in the mouth and pharynx. He probably had a vitamin deficiency which was rapidly overcome by the feedings; yet the difficulty persisted, although to a smaller degree. He emphasized the fact that his presentation was concerned mainly with the so-called functional cases rather than the organic. As for the nerve supply of the cricopharyngeus, the matter is not clear. Negus has admitted that he doesn't know. In some cases it is difficult to determine how the cricopharyngeus is affected in cases of unilateral paralysis of the larynx and pharynx. When the barium spills over into the larynx one cannot be certain whether it is due to diminished sensation or inability of the upper laryngeal mechanism to operate efficiently. Usually in the bulbar poliomyelitis cases the latter explanation is the more plausible.

Some of the organic causes were mentioned in the paper because they have to be ruled out before one can consider a diagnosis of functional disturbance. These can usually be demonstrated in good x-ray films.

About the voice changes in poliomyelitis, Dr. Lindsay could venture no opinion. However, in the case of scleroderma which he cited the patient did show a weakness of the laryngeal musculature which was bilateral, resulting in inability to close the glottis effectively. There was a definite air loss on talking, and the patient was unable to hiccough or laugh for the same reason.

The Globus Syndrome as Related to Demonstrable Pathology of the Cervical Spine.
Dr. Lewis D. Morrison.

Cases of globus in the absence of demonstrable pathology in the throat should not be dismissed as purely neurogenic and turned over to the psychiatrist. The psychiatric approach is too frequently abused. A careful history will often determine the mental equipment and attitude of the patient. Morrison has found that osteoarthritis is often overlooked in the x-ray examination and frequently is the real cause of the disorder. The x-ray findings are described by the author with appropriate illustrations. At times such symptoms as a grating sound in the neck on turning the head, tightness of neck muscle, and shooting pains may be elicited. Haphazard physical therapy may do harm, but heat and light massage are always helpful. X-ray therapy in small doses over a period of several weeks has proved beneficial; also corticotropin (ACTH), cortisone, gold therapy, and vitamins. In some cases esophagoscopy affords relief, and in a few instances surgery will have to be resorted to.

DISCUSSION

Dr. C. L. Jackson mentioned having recently had a patient with a condition closely resembling the last case cited by Dr. Morrison. He is convinced that we need to exercise greater care in the examination of x-ray films because all too frequently these alterations in the cervical spine are overlooked.

Dr. Louis H. Clerf stated that all too frequently we are likely to ascribe a lump in the throat to some functional disorder, which can be very unfair to the patient. On the other hand, a patient may have a lot of changes in the cervical spine and not have a lump in the neck. Certainly every patient with a complaint of a lump should have the benefit of a thorough study of the cervical spine with the help of a competent roentgenologist. Dr. Clerf showed a slide which indicated changes in two locations which undoubtedly were responsible for the trouble. However, since there was no indication for any serious intervention and since the patient was given to

understand the situation, she was content to carry on inasmuch as there was no tendency for the process to become aggravated.

DR. P. E. IRELAND asked the essayist about the influence of posture on this condition. He was thinking of high-heeled shoes and open toes, which orthopedic surgeons have long recognized as contributing to spinal curvature. It is also likely that a business executive sitting over a desk for hours at a time may be subject to this condition. Another possible cause is the posture when reading in bed. Mention was made of a collar for support. Dr. Ireland has fashioned one of a heavy felt, which the patient wears at home, especially when watching television, which, incidentally, he thinks is bad business. Isn't it likely that the difficulty in swallowing may be due not only to myositis or fibrositis but to changes in the bone?

DR. THOMAS C. GALLOWAY recalled an article in the French literature several years ago in which the thesis was developed that these cases are associated with a periarthritis, a process extending to the mucosa and involving the nerves in the upper portion of Auerbach's plexus. He inquired what Dr. Morrison thought was the pathology or pathogenesis of the lipping. Is the process limited to the articular end of the bone facets, or is it a process originating in the soft tissues which may involve some of the sympathetic and vagus fibers?

DR. ARTHUR W. PROETZ requested Dr. Morrison to elaborate his conclusions as to what the lump really is, whether an edema, a paresthesia, a myositis, or a muscle spasm. At times the sensation of a lump is absent entirely and might be termed functional. Yet it is not an imaginary thing. If one could really know the essence of the disturbance in the various structures mentioned that brings about the sensation of a lump, it would be easier to direct the patient and afford him relief.

DR. FREDERICK T. HILL ventured to add one more possible cause of this syndrome, namely, ossification of the stylohyoid membrane.

DR. MORRISON closed the discussion by answering the questions directed to him. Posture certainly is an important factor, and the collar is a useful adjunct in therapy. The important thing, however, is to alert the orthopedic men and the neurosurgeons to this condition, so they may take these cases off our hands. Posture such as the typist and the jeweler assume for long periods should be recognized as a factor. It should be noted that when fixation has lasted for a long time the pain may subside, which is why these cases don't worsen, as is the case with osteoarthritis. As for terminology, he objects to the term "globus hystericus" and prefers telling the patients that they have a cricopharyngeus muscle syndrome complex. Replying to Dr. Proetz, Dr. Morrison believes the symptom is due to a spasm of the cricopharyngeus muscle, which does not have to be continually present. Swallowing something will often relieve the condition, which is why nothing shows up in fluoroscopy. Warm foods are better than cold for relaxation. The article which Dr. Galloway referred to is fascinating reading. However, he believes the author has gone off the deep end a bit in interpreting the pathology. He was more impressed by an English article in which the author asked pathologists doing necropsies to investigate the tissues in this area, since grossly very little evidence of the true pathology has been available. The question is whether the pathology is in the bone itself, the ligaments, or the adjacent spaces into which calcium depositis may be laid down. He has requested surgeons doing a disk operation or a fusion of the spine to obtain enough tissue for histologic study. This plus good preoperative and postoperative x-ray studies might go far in supplying the answers.

The Clinical Significance of a Motor Pattern in Nucleus Ambiguus. DR. A. C. FURSTENBERG.

Experiments on monkeys by two different procedures have yielded considerable information on the distribution of the cells in the nucleus ambiguus controlling the action of the various muscles of the larynx. This knowledge may prove of great value in analyzing the complex manifestations of laryngeal dysfunction, which at times has been more or less baffling. The fact that the cells of origin for the cricothyroids, the abductor and adductor muscles have been identified could explain such phenomena as the loss of function of the true cords while the false cords maintain the function of closing and opening. It is therefore more than likely that isolated lesions centrally located are capable of producing bizarre combinations of laryngeal dysfunction where the activity of limited groups is affected. It may be assumed that since the pattern of localization has been demonstrated in the monkey it is logical to deduce that the motor pattern in the human being is more highly developed.

DISCUSSION

DR. LEWIS F. MORRISON could not agree with the statement that a single muscle or portion of a muscle is incapable of being injured or affected by surgery. The fact that the recurrent laryngeal nerve divides in the extralaryngeal spaces into as many as six branches makes it possible for only one branch to be damaged, thus affecting only the muscle which it supplies.

DR. JAMES M. ROBB commented on the accuracy and brevity of the presentation and suggested another field for investigation, namely, the innervation of the cricopharyngeus muscle.

DR. FURSTENBERG agreed, in reply to Dr. Morrison, that it is possible to affect a single muscle through one of the extralaryngeal fibers of the nerve. However, the complicated pattern of the nucleus ambiguus and the manifold fibers arising from its cells presents a most complicated arrangement, which accounts for the complex action of the various muscles which they innervate. Certainly the presentations of Drs. Lindsay and Morrison dealing with disturbed function point to the complex innervation referred to.

One slide which demonstrated degeneration of cells of origin of motor fibers of the recurrent larngeal nerve was examined by experts at Parke, Davis & Company who have been dealing with the effects of the viruses of poliomyelitis, and who confirmed without reservation the diagnosis of the type of degeneration one sees in the brain stem after section of the fibers.

The question of the nerve supply of the cricopharyngeus will sooner or later be definitely settled. Another item for further investigation relates to the difficulty of obtaining regeneration of the recurrent laryngeal nerve after end-to-end anastomosis or nerve graft. It is likely that it can be ascribed to the complicated pattern of the nucleus. The problem is to be able to follow the nerves in the process of regeneration all the way to their home base in the nucleus.

Blastomycosis of the Larynx. DR. WILLIAM C. THORNELL.

Dr. Thornell reports an unusual case of blastomycosis of the larnyx in a man of 44, a butcher. The first biopsy was inconclusive, tuberculosis being suspected but not proved. Streptomycin and dihydrostreptomycin failed to afford any relief. The cords became progressively thickened and partially obstructing. A second biopsy preceding electrocoagulation of the cords yielded a hyperplastic mucosa containing some giant cells and spheroid bodies with refractile walls which were identified as blastomyces. Some improvement followed the ingestion of iodides, but when the patient failed to maintain the therapy, the obstruction became more marked. Arytenoidectomy was performed on one side and then later on the other, affording considerable relief. With the resumption of iodide therapy the condition was brought to a standstill. Dr. Thornell suggests that the aromatic diamidines be employed in these cases, on the basis of some good reports following their use in blastomycosis in other areas. The case which he reported emphasizes the importance of bearing in mind the possibility of blastomycosis in any case of a granuloma of the larynx, once the commoner lesions such as tuberculosis and carcinoma are excluded.

DISCUSSION

DR. HOWARD W. D. MCCART showed slides of a case which was about to have a laryngectomy on the mistaken diagnosis of carcinoma. When viewed under low power it did strongly suggest carcinoma. However, the high power was able to show the marked hyperplasia. The condition cleared up entirely within a few weeks under stilbamidine, 150 mg.

DR. JAMES M. ROBB, commenting on the difficulty in diagnosing lesions of this sort, recalled the case of a woman of 40, who presented a small lump on the side of the neck which became red and tender. It was opened and drained and the material sent to the laboratory. Tuberculosis, histoplasmosis, and blastomycosis were ruled out. The pathologist suggested cat scratch fever, and comparison with a reprot on this entity in the *New England Journal of Medicine* proved it. It is called nonspecific regional lymph adenitis, and in this case was cured by potassium iodide.

DR. LEWIS F. MORRISON inquired whether the organism shown in the cells in the slide presented were still present at the time of the arytenoidectomy.

DR. THORNELL replied that the organisms were found in the tissue at the time of the arytenoidectomy even though the patient had had several courses of iodide therapy. It was difficult to keep the patient on the medication, but after complications developed he became more tractable and is reporting back frequently for observation.

Laryngeal Papillomata: Surgical Technique for Removal of Recurring and Obstructive Growths. DR. DEGRAFF WOODMAN.

Dr. Woodman described a case of recurring laryngeal papillomata in an adult, which was cured by a thyrotomy and resection of the tumor-bearing mucosa of the larynx, which involved both cords and ventricular area. An acrylic mold was placed in the larynx attached to the tracheotomy tube and left in situ for eight weeks. When it was removed the area operated on was seen to be completely healed and was covered with normal-appearing mucous membrane. One cord was immobile and the other had some adductor function. The voice was a hard whisper, adequate for the telephone; airway was sufficient and function of the upper part of the glottis was active.

Since the operation two growths have been removed from the trachea but the larynx has remained free.

The patient seemed to have a diathesis for mucosal pathology. She had undergone treatment for intramucosal carcinoma of the cervix and also was found to have papillomata in the bladder.

DISCUSSION

DR. EDWIN N. BROYLES stated that, generally speaking, thyrotomy is not indicated in these cases in children because of recurrences despite the surgery. However, if one is dealing with the adenotype of papilloma, one may be successful because this type shows less tendency to recur. In Dr. Woodman's case where the growths were present in the trachea, it seems doubtful whether the results would be final. He certainly hoped Dr. Woodman would get away with it. It is true that through the laryngoscope one is unable to do a submucous resection of the growths, which is the secret of complete removal. Therefore, the operation described by Dr. Woodman may have much more to offer.

DR. LEWIS F. MORRISON asked whether oxytetracycline (Terramycin) or chlortetracycline (Aureomycin) had been used preoperatively or postoperatively. They have in certain instances proved effective.

DR. MURDOCK S. EQUEN mentioned the case of a cheer leader who had been operated on a number of times in various clinics and finally developed a malignancy which necessitated a laryngectomy. It proves that these cases should not be taken lightly.

DR. FRED Z. HAVENS would not advise this procedure in children but did think that in adults where the condition is more amenable they might benefit by it. Reference has been made to possible malignant changes. A patient on whom he had operated at the age of 12 or 13 had growths in the trachea which were pronounced malignant. Yet the clinical course did not substantiate the diagnosis, since the patient kept having recurrences up to the age of 22 or 23, when last seen. A review of the earlier slides still classified the growths as malignant. Yet the patient is practically cured except for an occasional limited growth. The numerous operations led to a stenosis, which Dr. Havens plans to correct with a skin graft. In this case a thyrotomy proved the inadequacy of removal under endoscopy, because of the inaccessibility of nodules deeply situated in the ventricles.

DR. WOODMAN concluded by stating he had been hesitant about the procedure after hearing of failures elsewhere. However, the experience of many with implantation at the site of the surgical trauma impelled him to seek the more radical procedure in this case. Thus far the result has been good, it being already 14 months since the operation. She did have antibiotics at various times, without result.

Dr. Woodman recalled a discussion in which it was stated that removal of the growths followed by the application of Podophyllin had been successful in preventing recurrence.

Books

Cytology of Bronchial Secretions. By Dr. P. Hartmann. Paper, $5.70. Pp. 112, with 168 illustrations (many in color). Georg Thieme Verlag, Herdweg 63, (14a) Stuttgart N, 1955.

This book describes the cytologic examination of the bronchial secretions. This is subdivided into (1) the normal cells of the blood stream, (2) normal epithelial cells, (3) autolytic changes of the normal cells, (4) alveolar phagocytes and connective-tissue histiocytes, (5) atypical cells due to epithelial metaplasia, and (6) tumor cells. There is a chapter on the various methods of obtaining the various secretions, a chapter on fixation and staining, and, also, one on the diagnostic significance of cytologic findings. This book has an extensive bibliography and is excellent for the pathologist who can work in association with the bronchoscopist and thoracic surgeon.

The Bronchus and Tuberculosis. By Dr. A. Huzly and Dr. F. Bohm. Cloth, $13.55. Pp. 138, with 250 illustrations (many in color). Georg Thieme Verlag, Herdweg 63, (14a) Stuttgart N, 1955.

This excellent book graphically describes the pathologic findings in the bronchus as seen in tuberculosis of the lung. The involvement of the bronchus both extrinsically and intrinsically is beautifully illustrated. Bronchograms with descriptive line drawings amply clarify the underlying pathology. This pathology is shown in relation to (1) tuberculosis of the pulmonary parenchyma; (2) endothracic lymph node tuberculosis; (3) tuberculosis of the pleura; (4) bronchoscopic findings as a result of active therapy for pulmonary tuberculosis, namely, the findings following collapse therapy, phrenic nerve avulsion, and pulmonary resection; (5) true tuberculous involvement of the bronchi; (6) the course of bronchial tuberculosis; (7) the problems involved, both physiologic and therapeutic, in the treatment of resolution, fibrosis, and scar-tissue formation of the bronchus. Numerous case reports have been included to illustrate the many points of discussion. In addition to the usual pathologic findings, therapy is discussed and the results following therapy are clearly indicated throughout the discussion. This is a well-organized book, and it is extremely valuable for both the phthisiologist and the bronchoscopist.

Salivary Gland Tumors. By Donald E. Ross, M.D., F.A.C.S., F.R.C.S. (Edin.); Diplomate, The American Board of Surgery, Chief Surgeon, Ross-Loos Medical Group, Los Angeles. Price, $7.50. Pp. 96, with 47 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1955.

While the submaxillary and sublingual glands are sufficiently covered, chief attention is given to the parotid, which so long has been a *noli me tangere* for general surgeons. "Physicians do not realize adequately the serious nature and the potential dangers of malignancy in these tumors." Dissection of the parotid is difficult, and one is always in fear of facial nerve paralysis. One object of this book is to go intensively into the anatomy and pathology, so that the surgeon will have well in mind definite criteria for surgery. The author states that the facial nerve can usually be spared if careful surgical technique is carried out. Chapter XI deals with this item in extenso. As for radiation as a substitute for surgery, the author seems to vary between "yes" and "no." He says that preoperatively "we believe it to be of little value." However, in the tumefaction of children (lymphadenopathy) radiation "should be tried before submitting the patient to a hazardous operation." And in Mikulicz' disease, "many of our cases have been greatly benefited by radiation."

This book is a comprehensive and intensive study of a disfiguring disease which may be malignant although thought to be benign in most instances. There are nearly 400 references to the literature and many figures in black and white. Also, one finds a few excellent plates in color. One would hardly undertake removal of the parotid unless well prepared by cadaver work. A reading of this text before dissecting will repay the reader well for his time and effort. It is an excellent book.

Auditory Disorders in Children. By Helmer R. Myklebust. Price, $6.00. Pp. 356, with 23 tables and 5 figures. Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1954.

This book presents procedures for differential diagnosis of auditory disorders in children below the age of six years. The author stresses the importance of considering a hearing disorder in relation to the child's total developmental behavior.

There are five parts to the book, including an introduction and a series of case studies from the author's aphasia clinic. Part 2 describes history taking and evaluating the information in terms of a final diagnosis. Part 3 elaborates on peripheral deafness, aphasia psychic deafness, and mental deficiency, with behavior differences characterizing each condition. The fourth part describes examination methods. The author emphasizes informal observations over formal audiometric tests. Psychological examination is of the utmost importance and is actually the basis of the author's differential methods. The approach is primarily through the eyes of a psychologist. There is much information throughout the book, and it is a worth-while contribution to this confusing aspect of otology.

1955 Medical Progress. Edited by Morris Fishbein, M.D. Price, $5.00. Pp. 346. The Blakiston Company (Division of McGraw-Hill Book Company, Inc.), 330 W. 42nd St., New York 36, 1955.

This book is a review of advances in medicine as reported through the year 1954. It is a collection of significant papers written throughout the year and compiled by outstanding authorities in each particular field of medicine.

The subdivisions include gastroenterology; surgery; obstetrics; cardiovascular diseases; psychiatry; allergy; diabetes; rheumatic diseases; diseases of the chest; nutrition; dermatology; ophthalmology; orthopedic surgery; infectious diseases; physical medicine; liver disease; endocrinology; ear, nose, and throat; new drugs, and medicine.

For the general practitioner and the busy specialist, the book presents a considerable number of interesting subjects, with the most recent interpretation, both from the standpoint of diagnosis and treatment. Each subdivision has its own detailed bibliography so that a ready reference can be made to the original article and its source and publication.

Anatomie radiographique du massif facial (Radiographic Anatomy of the Face). By Maurice Bouchet, Otolaryngologist to the Hospital of Paris, and George Dulac, Electro-Radiologist and Senior Chief of Service of the Curie Foundation. Stitched, 2800 francs; linen, 3600 francs. Pp. 188, with 103 illustrations. Masson & Cie, 120 boulevard Saint-Germain, Paris, 6e, 1955.

The authors present a graphic description of the various procedures employed in roentgenograms of the face and sinuses. They depict graphically with line drawings, schematic sketches, and actual roentgenograms both the conventional and tomographic procedure to visualize the facial structures and sinuses. The explanatory notes are extremely concise and descriptive. This comprehensive book should be of extreme value to the roentgenologist and also a valuable aid to the rhinologist who prefers to interpret the roentgen films. Considerable emphasis is placed on the value of tomography. The last chapter deals with interpretations in which both roentgenograms and line drawings are presented so that each anatomic structure can readily be identified. In addition, there is an extensive bibliography covering all the problems that are reviewed in the text.

Hearing Therapy for Children. By Alice Streng, M.A.; Waring J. Fitch, M.A.; LeRoy D. Hedgecock, Ph.D.; James W. Phillips, M.D., and James A. Carrell, Ph.D. Price, $6.75. Pp. 371. Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1955.

This is an excellent contribution to literature of the problems of the hard of hearing child. It is written by authors who are outstanding specialists in their various endeavors. The chapters include a brief history of deafness, the problems of the deaf child, medical causes of hearing loss and treatment, audiometry and case findings, clinical audiometry, hearing aids for children, educational aspects of hearing loss, and the difference in education between the mild and the

moderately deaf child, as well as the education of a child with severe and profound hearing loss. In the appendix is an excellent but incomplete list of the established hearing centers in the United States. The book has considerable usefulness for school nurses and lay people interested in doing audiograms on school children. Of particular interest is the specific information on hearing therapy for children and the educational aspects of hearing loss. The otologist who does work with hard of hearing child will benefit greatly by digesting this most interesting book.

Diseases of the Ear, Nose, and Throat. By William Wallace Morrison, M.D. Second Edition. Price, $10.00. Pp. 756, with 638 illustrations. Appleton-Century-Crofts, Inc., 35 W. 32nd St., New York 1, 1955.

This edition shows considerable revision to include many new developments and more compact presentations. It is like the first edition. It is extremely well organized, particularly for teaching undergraduates and postgraduates and for use by the general practitioner. Emphasis is placed upon important positive information. Obsolete and unproved concepts and therapeutics are disregarded. Stress is given to common conditions encountered in practice. The diagrams are drawn by the author so as to clarify the text and show early stages rather than late stages of pathology. This enables the reader to recognize disease much earlier, when it is more responsive to therapy. This book is an outstanding contribution in its revised form.

Peripheral Circulation in Man (A Ciba Foundation Symposium). By G. E. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Jessie S. Freeman, M.B., B.S., D.P.H.; assisted by Joan Etherington. Price, $6.00. Pp. 219, with 72 illustrations. Little, Brown & Company, 34 Beacon St., Boston 6, 1954.

Bibliography on Hearing. By the Harvard University Psycho-Acoustic Laboratory. Price, $7.00. Pp. 557. Harvard University Press, Cambridge 38, Mass., 1955.

This volume is a must for all scientific libraries and will be a boon to persons interested in research on problems of hearing. It will also be a great time saver for both investigators and clinicians interested in literature on any aspects of hearing. The book covers the literature of the world, including Slavic references not often located. The bibliography of 557 pages includes books and articles of historical as well as current interest and is arranged alphabetically by authors. Foreign references are quoted in English. German and French references are also listed with original titles. Another section contains a classification by subject under 315 headings.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. D. S. Gorrell, 110 Medical Arts Building, Calgary, Alta.

Secretary: Dr. G. Arnold Henry, 328 Medical Arts Building, Toronto.

Place: Chateau Frontenac, Quebec, Canada. Time: June 6-7, 1956.

International Bronchoesophagological Society

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.

Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

Meeting: Fourth International Congress of Bronchoesophagology.

President of Congress: Dr. Antonio Carrascosa.

International Congress of Audiology

Place: Leyden, Netherlands.

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Juan Manuel Tato, Azcuenaga 235, Buenos Aires, Argentina.

Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

Meeting: Fifth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.

Place: San Juan, Puerto Rico. Time: April 8-12, 1956.

President of Congress: Dr. Juan H. Font, Medical Arts Building, San Juan.

General Secretary: Dr. C. E. Muñoz MacCormick, P. O. Box 9111, Santurce 29, Puerto Rico.

Sixth International Congress of Otolaryngology.

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.

General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.

Place: Hotel Statler, Washington, D. C. Time: May 5-10, 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. John R. Lindsay, Chicago.

Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.

Place: Chicago. Time: June 11-15, 1956.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Algernon B. Reese, 73 E. 71st St., New York 21.

Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave., Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN BOARD OF OTOLARYNGOLOGY

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.

Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

Place: Sheraton-Mount Royal Hotel, Montreal, Canada. Time: May 6-11, 1956.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Daniel S. Cunning, 115 E. 65th St., New York 21.

Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.

Place: Sheraton-Mount Royal Hotel, Montreal, Canada. Time: May 15-16, 1956 (afternoons only).

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Bernard J. McMahon, 8230 Forsyth Blvd., Clayton 24, Mo.

Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.

Place: Seigniory Club, Montreal, Canada. Time: May 13-14, 1956.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, Iowa City.

Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

Place: Sheraton-Mount Royal Hotel, Montreal, Canada. Time: May 15-17, 1956 (mornings only).

SECTIONS:

Eastern.—Chairman: Dr. John A. Murtagh, Hanover, N. H.

Southern.—Chairman: Dr. J. M. Robison, Houston, Texas.

Middle.—Chairman: Dr. William H. Craddock, Cincinnati.

Western.—Chairman: Dr. David D. De Weese, Portland, Ore.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. William J. McNally, 1509 Sherbrooke St. West, Montreal 25, Canada.

Secretary-Treasurer: Dr. Lawrence R. Boies, 90 S. Ninth St., Minneapolis 2.

Place: Seigniory Club, Montreal, Canada. Time: May 11-12, 1956.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Joseph Gilbert.

Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

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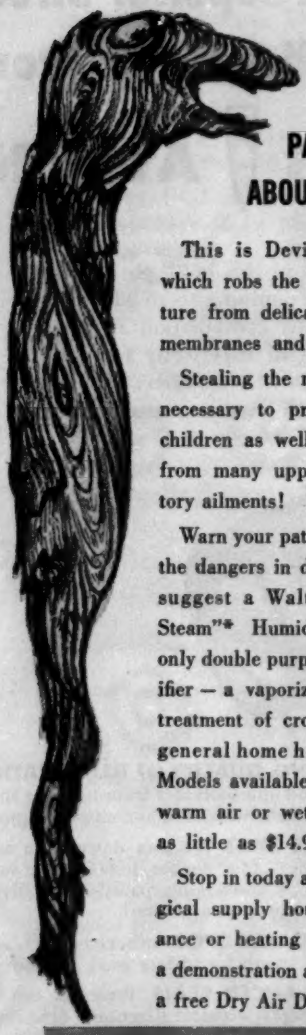

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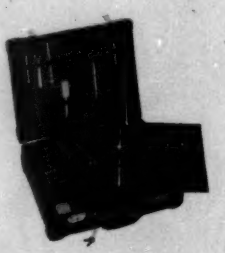

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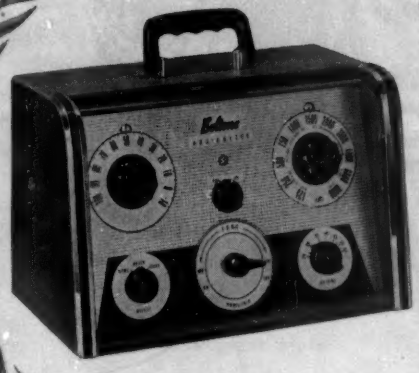

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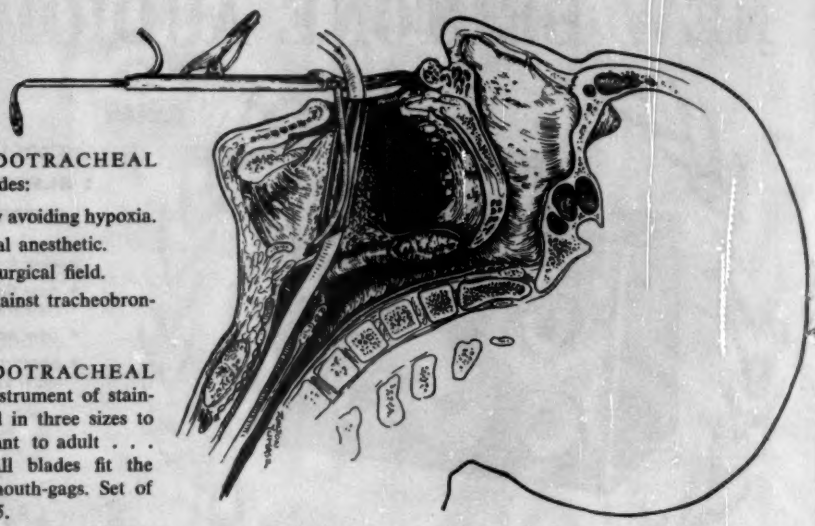

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* Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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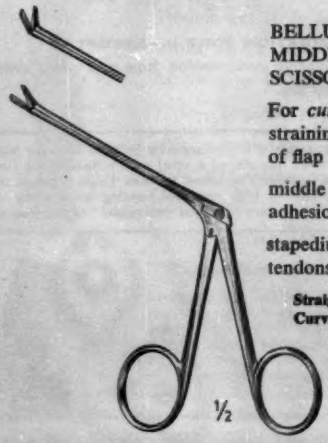

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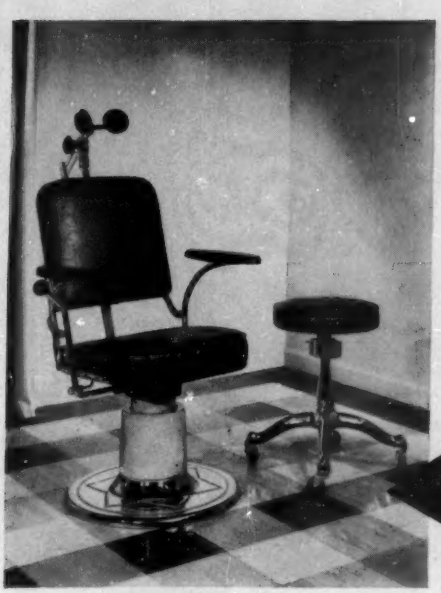

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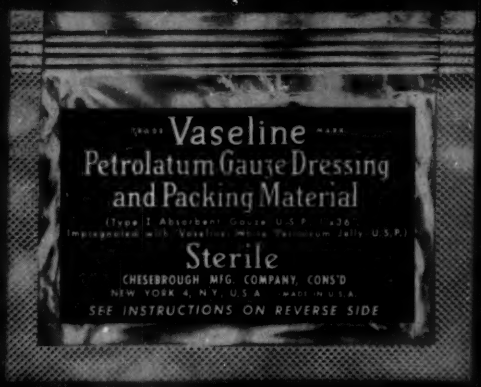

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2. Yarlett, M. A., Gershenfeld, L., McClenahan, W. S.: Drug Standards 22:205, 1954.

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